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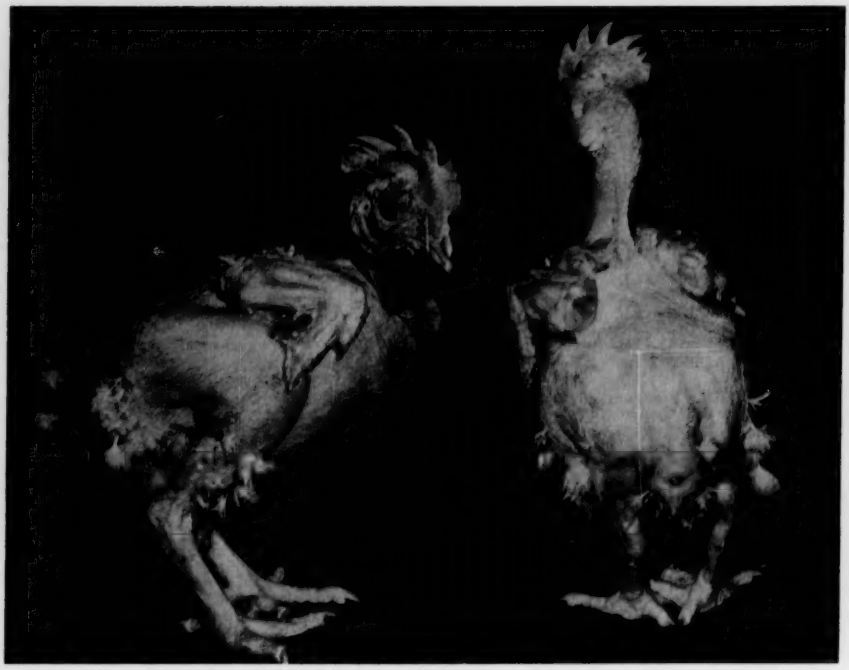

SCALELESS—A RECESSIVE MUTATION

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Duplicated Linkage in Cotton

Colchicine Induced Sorghum Mutants

A Nutritional Strain Difference in Mice

Chromosomal and Cytoplasmic Microfibrillae

VOLUME 48 MARCH-APRIL, 1957 NUMBER 2

The American Genetic Association

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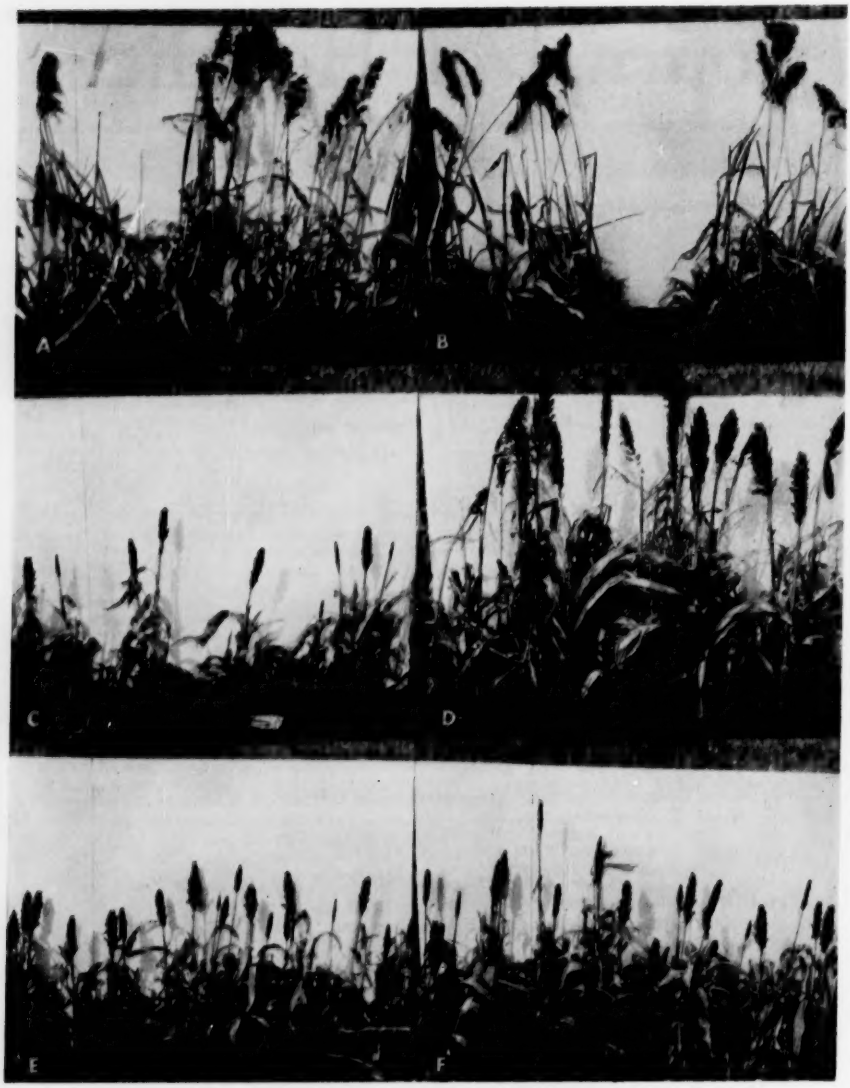


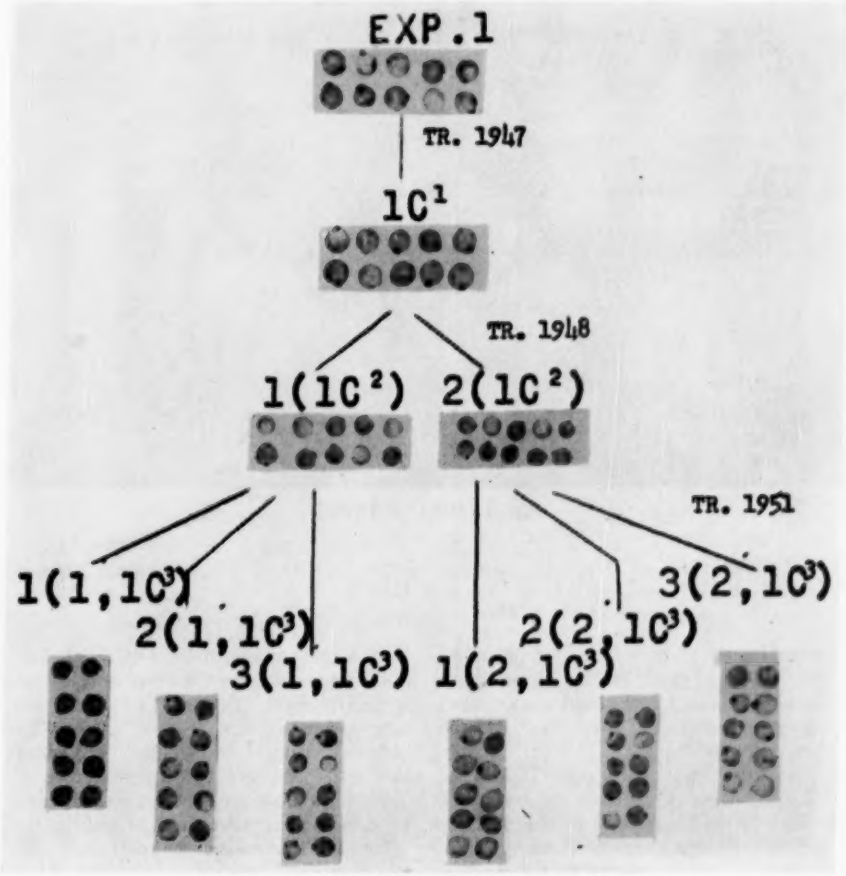
THREE GENERATIONS OF NEW MUTANTS

Figure 1

A—Shows the plant types for the original variety Experimental 1. Three successive generations of new mutants are shown in *B-F*. *B*—1C^1; *C*—1(1C^2); *D*—1(1,1C^3); *E*—2(1,1C^3); *F*—3(1,1C^3). Each generation was derived from the preceding generation by colchicine treatment.

A LINEAL SERIES OF MUTANTS INDUCED BY COLCHICINE TREATMENT

C. J. Franzke and J. G. Ross*



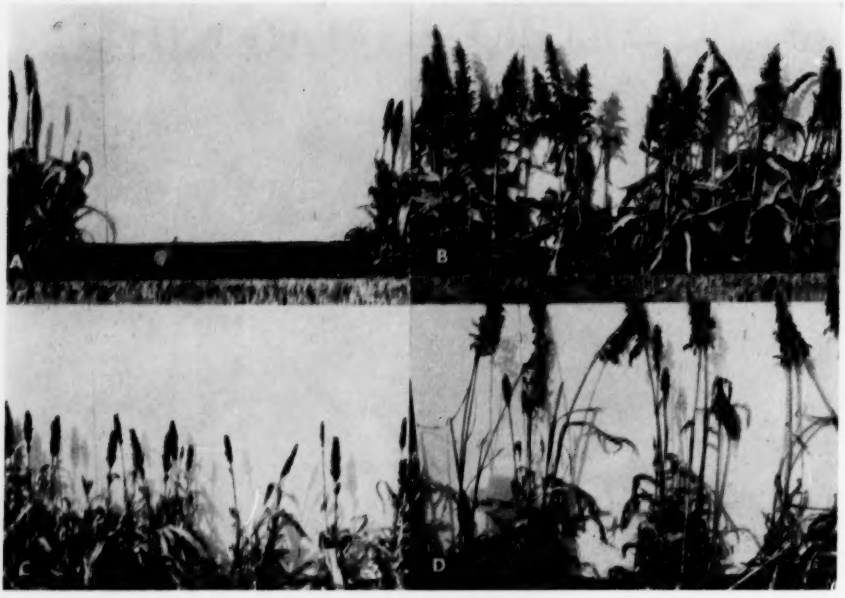
ORIGIN OF COLCHINE TREATED MUTANTS

Figure 2

The diagram shows the derivation by colchicine treatment of each true-breeding mutant from the preceding mutant. The first digits represent plant numbers while superscripts indicate the number of successive treatments in the pedigrees of the mutants. Seeds from each are about natural size.

TRUE-BREEDING mutants in sorghum induced immediately by colchicine treatment from the variety Experimental 3 have been reported previously[2,4]. Since Experimental 3 had in its ancestry Sudan grass and forage sorghum, and since some of the new types obtained by colchicine treatment

*Agronomy Department, South Dakota State College, Journal Paper Number 368, South Dakota Agricultural Experiment Station, Brookings, South Dakota. This investigation was supported in part by research grant G-2217 from the National Science Foundation.



FOUR MUTANT TYPES

Figure 3

A—Plant types are shown for the second mutant, $2(1C^2)$. *B-D*—Shows three mutants, $1(2,1C^3)$, $2(2,1C^3)$ and $3(2,1C^3)$ derived from $2(1C^2)$.

resembled these, it was at first proposed that masked gene blocks from these sources may have been uncovered through reductional grouping of the chromosomes in the somatic tissue. However, it was shown by Harpstead *et al*[3] that no detectable relocation of chromatin had occurred in the mutants and that the new characteristics appeared to have arisen from chromatin changes of the nature of point mutations.

This study is concerned with colchicine treatment of a different sorghum variety, Experimental 1, and with retreatment of mutants thus induced, i.e., it deals with the production of lineal series of new mutants each of which was immediately true-breeding.

Materials and Methods

Experimental 1, the sorghum variety used in this investigation, originated from a cross made in 1939 at South Dakota State College between a selection from Grahoma, (36"-42" in height) and a true-breeding line (30"-40" in height) obtained from Hays, Kansas. The latter line had been selected from a cross of Dwarf Feterita and Dwarf Freed, both of which were the same height as the selection. The method of treatment with colchicine has been reported previously[2]. For simplification, the series of mutants derived from this variety by colchicine treatment and retreatment have been given special designations as shown in Figure 2. The mutant arising from colchicine treatment of Experimental 1 is designated as $1C^1$; mutants arising from retreatment of this mutant, as $1(1C^2)$ and $2(1C^2)$; and mutants arising from a third treatment as $1(1,1C^3)$, $2(1,1C^3)$ etc., or as $1(2,1C^3)$, $2(2,1C^3)$, etc. depending on the mutant treated. Thus

TABLE I. Days to pollination and height of Experimental 1, and mutants derived from repeated treatments 1952-1955

Line Designation	Origin	Days to pollination					Height in inches					Seed Wgt.†
		1952	1953	1954	1955	Mean	1952	1953	1954	1955	Mean	1955
Exp. 1	*	54	62	63	55	58.5	56	62	51	49	54.5	2.78
$1C^1$	Exp. 1 trtd. 1947	54	60	57	55	56.5	60	63	54	52	57.2	2.91
$1(1C^2)$	$1C^1$ trtd. 1948	63	71	75	63	68.0	30	30	25	28	28.2	1.71
$1(1,1C^3)$	$1(1C^2)$ trtd. 1951	72	87	69	55	70.8	40	39	47	47	43.2	1.87
$2(1,1C^3)$	$1(1C^2)$ trtd. 1951	86	84	67	63	75.0	31	32	40	31	33.5	1.71
$3(1,1C^3)$	$1(1C^2)$ trtd. 1951	83	71	78	65	74.2	27	31	27	32	29.2	1.44
$4(1,1C^3)$	$1(1C^2)$ trtd. 1951	80					30					
$5(1,1C^3)$	$1(1C^2)$ trtd. 1951	80					27					
$6(1,1C^3)$	$1(1C^2)$ trtd. 1951	65					27					
$7(1,1C^3)$	$1(1C^2)$ trtd. 1951	76					27					
$2(1C^2)$	$1C^1$ trtd. 1948	72	70	80	65	71.8	25	28	23	28	26.0	1.10
$1(2,1C^3)$	$2(1C^2)$ trtd. 1951	59	64	68	59	62.5	38	40	38	35	37.8	1.90
$2(2,1C^3)$	$2(1C^2)$ trtd. 1951	76	72	82	65	73.8	28	27	22	29	26.5	1.47
$3(2,1C^3)$	$2(1C^2)$ trtd. 1951	49	70	49	50	54.5	39	33	47	47	41.5	2.24
$4(2,1C^3)$	$2(1C^2)$ trtd. 1951	72					27					
$5(2,1C^3)$	$2(1C^2)$ trtd. 1951	70					27					
LSD at 5% level						9.4					11.2	0.10

*Origin described in text. †Mean weight in grams of three samples of 100 seeds each.

the third mutant derived from the second mutant which had been derived from the original mutant is designated as $3(2,1C^3)$.

Observations and Discussion

The series of mutants obtained from Experimental 1 by repeated colchicine treatments is diagrammed in Figure 2, which also includes illustrations of the seed size of each mutant. Plant types of the mutants are illustrated in Figures 1 and 3. Mutant $1C^3$ (Figure 1) was derived by colchicine treatment in 1947. This new type was slightly taller, but not significantly so (Table I), had approximately the same maturity, and produced seeds somewhat larger than the original (Figure 2). When this mutant was again treated with colchicine in 1948, two mutants, $1(1C^2)$ and $2(1C^2)$ were obtained. These mutants are very similar in general plant type, both having a tight, narrow panicle termed rat-tail (Figures 1 and 3). The most obvious differences are that $1(1C^2)$ has slightly larger seeds (Table I) and its seed is much more viable; stands of $2(1C^2)$ are always poor as shown in Figure 3. The true-breeding nature of Experimental 1, of $1C^1$, and of $1(1C^2)$ has previously been described[4].

Seven seedlings of $1(1C^2)$ and five seedlings of $2(1C^2)$ were treated with colchicine in 1951, and selfed progenies were grown from these in 1952. Maturity, as measured by days to pollination, and height were observed on these progenies and are reported in Table I. Progenies of four treated plants of $1(1C^2)$ and of two treated plants of $2(1C^2)$ were discarded since no obvious difference from the original untreated rat-tail type could be detected. Therefore, data were not collected on progenies from these six plants for the years 1953-55. Progenies of the other plants, however, were different from their untreated parents for four succeeding generations, providing evidence that mutations had resulted from the colchicine treatment. The most outstanding differences are found in $1(1,1C^3)$, $1(2,1C^3)$ and $3(2,1C^3)$, as illustrated in Figures 1 and 3. Significant differences for maturity and/or height in comparison with the immediate parents are indicated in Table I. The mutant $3(2,1C^3)$ appears superficially very similar to the original variety, Experimental 1, while $1(2,1C^3)$ and $1(1,1C^3)$ are quite unlike that variety. Some differences such as restored viability in $2(2,1C^3)$ were noted in the field between each of the other lines and their immediate parents, though measurements of maturity and height were not significantly different. All 10 lines were found to be diploid.

This study indicates that, following colchicine treatment, mutant lines may arise which are quite different from any known ancestor of the treated material. Mutant $3(2,1C^3)$ in this investigation, like many of the mutants induced by colchicine treatment during previ-

ous investigations with Experimental 3,[2] resembles a type known to be in the pedigree of the treated line. However, the appearance of distinctly new phenotypes, like the rat-tail types, provides evidence that such a resemblance is not necessary. These results are consistent with the later hypothesis suggested for the induction of mutant plants in Experimental 3, namely that the new types arise as the result of multiple point mutations[1,3], and not with the earlier hypothesis, that the mutants might result from the unmasking of gene blocks[2]. However, assuming colchicine to be an agent for gene mutation is not sufficient to explain the immediate true-breeding nature of the mutant diploid plants, since it would be necessary to assume multiple identical mutations at identical loci on both homologues; such would not occur if our present concept of the nature of the gene is correct. A reductional division in the somatic tissue subsequent to gene mutations and followed by doubling of the chromosomes would produce true-breeding lines. Gene mutations both prior and subsequent to a somatic reductional division and chromosomal doubling would result in a mutant line true-breeding for some characteristics but segregating for others. Such mutant lines have been observed (unpublished).

Summary

A sorghum variety, derived by selection from crosses of known grain sorghums, was subjected to colchicine treatment. A slightly taller, true-breeding line was obtained which, when retreated, gave two dwarf mutants which also bred true immediately and which were distinctly different from any types known to be in the pedigree. Retreatment of these two mutants gave new true-breeding types, some of which resembled the original untreated variety. Since new diploid forms arise which are not known in the ancestry, this study provides further evidence that colchicine is an agent for gene mutations. However, the immediately true-breeding nature of these lines appears to be most easily explained by a reductional division of the chromosomes in the somatic tissue with subsequent restoration to the diploid number.

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CHROMOSOMAL AND CYTOPLASMIC MICROFIBRILLAE IN SPERM OF AN ICERYINE COCCID

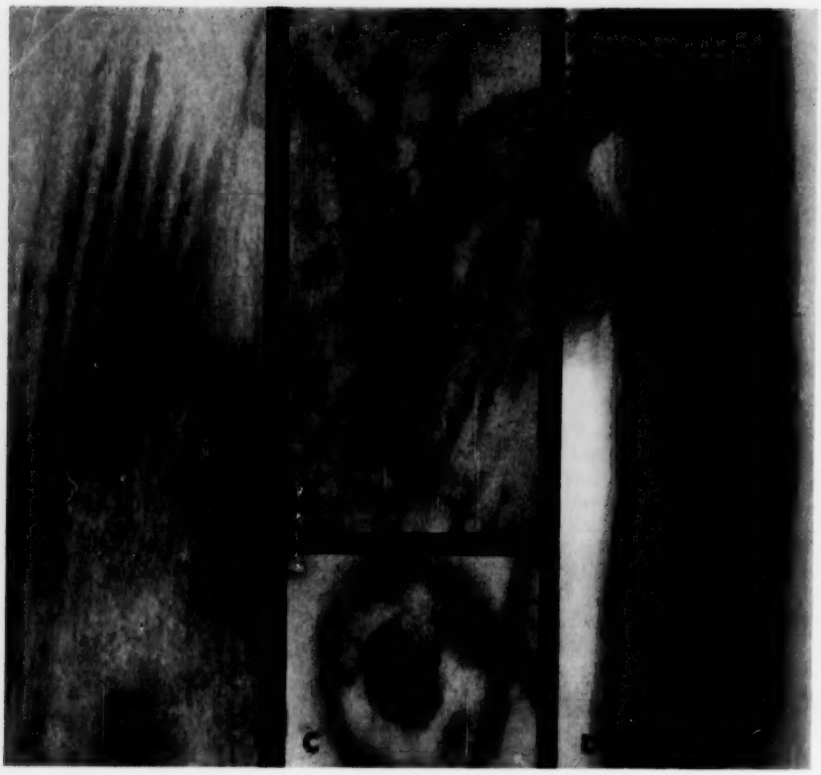
BERNARD R. NEBEL*

THE extensive investigations concerned with salivary gland chromosomes of many species of Diptera have perhaps made it sufficiently clear that polytene chromosomes do exist. To what degree polyteny is a general property of all chromosomes is not known. Among electron microscopists, Ris[7] and Kaufmann[4] have made specific statements. According to Ris[7] the microfibrillar unit is very similar in *Triturus, Tradescantia, Lilium* and the rat. In shadowed material the microfibril, which has a diameter of about 200 A, is a coiled cylinder with a hollow core. The wall of the cylinder is about 50 A thick. The core is presumably DNA which is poorly fixed in osmium tetroxide. Kaufmann[4] describes the chromatin in the helically arranged chromonemata in prophasic nuclei of *Tradescantia* stamen hairs. He states that he has seen "64 identifiable subsidiary strands assumedly arranged as intertwined pairs to form a hierarchy of pairs of pairs." Like Ris, Kaufmann considers that the basic unit is a hollow cylinder with a wall approximately 40 A thick. The wall consists of "another pair of coiled threads surrounding a central axis 40 A in diameter."

Microfibrillae in *Helix* were demonstrated by Grassé *et al.*[1, 2] However, in *Tradescantia, Helix* and in most other metazoa it is difficult to establish specifically a definite numerical relationship between individual chromosomes and the respective number of constituent microfibrillae. Even in their latest paper Grassé *et al.*[2] go to great length in describing the microfibrillae of *Helix* through successive stages of spermatogenesis without being able to determine how many "chromonemata" belong to an individual chromosome. They describe the width of their microfibrillae which they call "chromonemata" as varying between 70 and 90 A. They give the total length of a single thread as 230-240 μ. At the stages they observed in *Helix pomatia* the territory of individual chromosomes cannot be seen. Hence the number of microfibrillae per chromosome was not determined in *Helix*.

In marked contrast in the sperm of the iceryine coccid *Steatococcus tuberculatus* a single chromosome is distinctly defined. Hughes-Schrader[3] has shown the development of the sperm of *Steatococcus* to be as follows: The spermatids are binucleate. Each nucleus with the aid of the common cytoplasm leads to the formation of a separate spermatozoon. The nucleus approaches the cell margin and forms a small cone-shaped protrusion or papilla. Around this papilla the cytoplasm builds a larger cone-shaped cytoplasmic collar and cup. Beyond this cone-shaped collar and cup the cytoplasm builds the extended sheath of the sperm, a long hollow cylinder. Into the lumen of this cylinder the two chromosomes of a *Steatococcus* spermatid nucleus migrate in tandem. Within the sperm sheath there is at no time an overlap of individual chromosomes. The sperm is composed of the two chromosomes of the haploid set extended in linear, serial alignment and enveloped in a thin cytoplastic sheath (Hughes-

*Division of Biological and Medical Research, Argonne National Laboratory, Lemont, Illinois. The author wishes to express his appreciation to Dr. Sally Hughes-Schrader who not only suggested and supplied the material for this study but also furnished much valuable guidance and encouragement throughout. Dr. Hughes-Schrader, Dr. F. Wasserman and Dr. E. L. Roth critically reviewed the manuscript. Miss D. H. Papierski and Mr. O. T. Minick lent intelligent, critical and constructive technical help and scientific understanding.

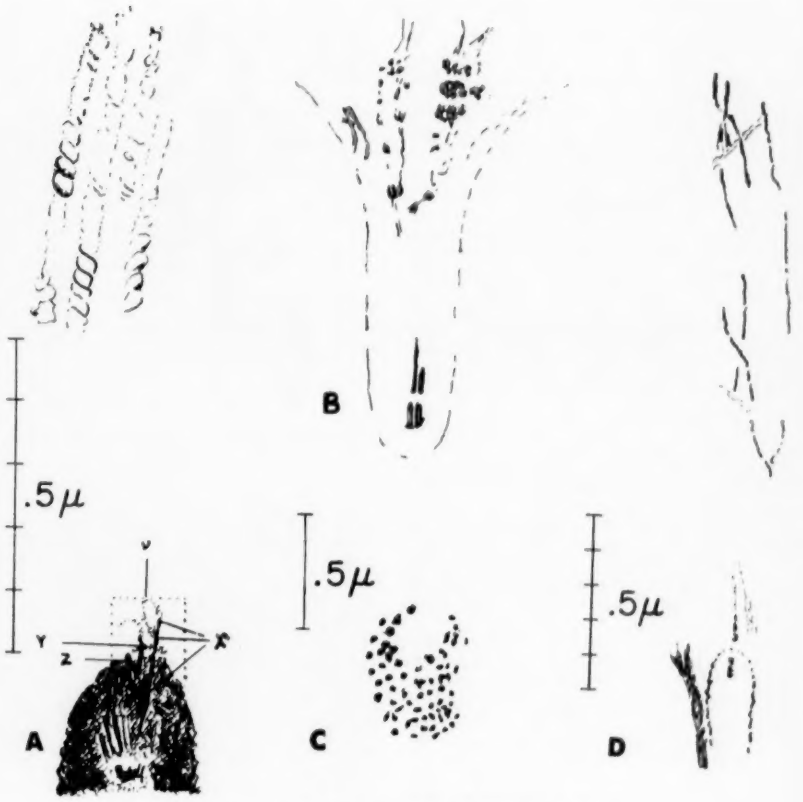


ELECTRON PHOTOMICROGRAPHS OF A STEATOCOCCUS CHROMOSOME

Figure 4

A—Oblique section of a iceryine coccid spermatozoon. Note cytoplasmic microfibrillae, at upper end of figures, representing the sheath of the spermatozoon. At lower center is seen the profile of the chromosome in the axial region of the spermatozoon. At the upper margin of this profile chromosomal microfibrillae are present. Figure 5*A* emphasizes the salient feautres of Figure 4*A*. × 92,000. *B*—Near sagittal section through the cytoplasmic "collar and cup" with cytoplasmic microfibrillae in oblique profiles and the central (upper) ostium through which chromatin is passing in two visible major central strands and apparently also in active parietal streaming into the sperm sheath proper. × 32,000. *C*—Shows a "tail" in cross section with about 64 microfibrillae grouped into two areas suggesting chromatids. The stage is early sperm before bundles are formed. × 32,000. *D*—A longitudinal profile of a spermatozoon with prominent chromosomal microfibrillae at all levels. The 30 A chromosomal microfibrillae shown in side view are seen in cross section in *C*; variations in their arrangement within the respective tail sheaths are believed to result from differences in developmental stage and differences in treatment, e.g. fixation and cutting angle; the stage is likewise early sperm, before bundles are formed. × 50,000.

Schrader[3]). Thus a single *Steatococcus* chromosome can be sectioned and both the structure of the microfibrillae and the number of the components may be investigated. For this reason Dr. Hughes-Schrader suggested the use of sperm of this iceryine coccid for electron microscopy in the present study.



ELECTRON MICROGRAPH DETAILS

Figure 5

This drawing has been prepared to enable the reader to find the essential parts in the electron photomicrographs. For explanation see legend for Figure 4.

Materials and Methods

The material, *Steatococcus tuberculatus* Morrison, was furnished by Dr. S. Hughes-Schrader. It was imported under special permit from the U. S. Department of Agriculture and Plant Quarantine and maintained in a greenhouse culture at the Department of Zoology at Columbia University. Fixation was carried out under the supervision of Dr. Hughes-Schrader at Columbia University.

Testes were dissected from late 3rd and early 4th instar males under a binocular microscope placed under a hood to protect the investigator from osmic acid fumes. Dissection was started in air and completed in a generous drop of 1 percent osmium tetroxide adjusted with Michaelis Veronal buffer to pH 7.4. Subsequent dehydration and embedding was completed within 24 hours. The medium was a mixture of 40 parts ethyl methacrylate and 60 parts butyl methacrylate. 250-A sections were cut on a Porter-Blume microtome and mounted on thin pure carbon membranes, from which the colloidion had

been removed with absolute alcohol.

The instrument used was a RCA EMU 3A electron microscope with a 25-μ objective aperture and a 100-kv beam. The original negative magnifications were, Figure 4*A*: × 22,000; Figure 4*B* and *C*: × 8,500; Figure 4*D*: × 12,000.

Description

Since electron photomicrographs of structures near the limits of resolution can be evaluated only after many pictures are taken, drawings have been added to point up the salient features of the photomicrographs. Most laboratories report a productivity ratio of about 500:1 for pictures taken over pictures published. This ratio is probably conservative compared with the observations made with light microscopy.

Figure 4*A-D* shows the original photographs and Figure 5*A-D* shows the line drawings made from the photographs to facilitate observation. In the case of Figure 6 this procedure has been carried one step further. An original pair of the photographs of the through-focus series were viewed and critically compared under a binocular dissecting scope, and a freehand drawing was made of the microfibrillae within the small area indicated in Figure 5*A*.

Drawings are rarely used in electron microscopy. This is unfortunate for a number of reasons well known to artists and illustrators. The human eye and visual cortex form a system which selectively amplifies messages pertaining to patterns. This task is as yet not equalled by any mechanical device. To appreciate an unfamiliar pattern requires learning. Thus first observations of microfibrillae are labored but with learning their presence grows readily obvious. Any pattern is best communicated to the novice in a drawing free of confusing detail (Nebel[5]).

This removal of irrelevant detail does not in any sense change the limits of resolution. Below 40 A extreme caution must be exercised, and below 15-20 A interpretation should not be attempted. At the upper limits of photographic enlargement, 20 × the original, grain is a prominent part of the image. These pitfalls can be avoided by careful comparison of the corresponding images in a through-focus series and by viewing many different micrographs of the same structure. All these precautions were vigorously applied in drawing Figure 6.

Figures 4*A* and 5*A* show an oblique section of a spermatozoon from a testis fixed approximately seven days after the second molt. As has been shown by Hughes-Schrader[3] the two chromosomes of each spermatid nucleus migrate in tandem into the tail in order to complete the spermatozoon. There is no sperm head.

CHROMOSOMAL MICROFIBRILLAR UNITS

Figure 6

This is an interpretive drawing of the small rectangle shown in Figure 4*A*. This area of the original photographs was viewed under a binocular dissecting microscope and drawn with the aim of avoiding artifacts due to photographic grain and electron optical illusions such as scatter. The area was drawn electrically to avoid confusion. V, Y and Z of Figure 4 are shown here but X was omitted. Thus this represents a typical group of 30 A chromosomal microfibrillar units. Any parts of these structures measuring less than 15 A in diameter are not considered cogent evidence. Approximately 1.5 × 10^5.

The cytoplasm of the spermatid forms the collar and cup, cf. Nebel[6], which under electron microscopy in sagittal section appears as a heavy walled funnel (Figures 4*B* and 5*B*). The tailcap apparently organizes the cytoplasmic sheath of the spermatozoon. This sheath in the region of the shaft, not at the ends, consists of about 115 parallel cytoplasmic microfibrillae arranged usually in three tiers (Nebel[6]). Thus it forms a hollow cylinder, seen in cross-section in Figure 4*C*, in which the two chromosomes lie in tandem, without overlap. The chromatin is surrounded by a moderate amount of karyolymph, which seems to occupy a fairly distinct peripheral position in Figure 4*C*, but may be more diffusely disposed as in Figure 4*D*, a longitudinal profile of the shaft region of the sperm. In Figures 4*A* and 5*A* the dense chromosomal axis, obliquely cut, is seen in the lower central part of the figure.

Cytoplasmic microfibrillae. The cytoplasmic microfibrillae of the tail sheath are seen in the upper part of Figures 4*A* and 5*A*. They

resemble rather tightly coiled straight extended parallel ropes each consisting probably of two component strands. The direction of coiling is occasionally reversed. The diameter of each compound fiber is 150-200 A.

Chromosomal microfibrillae. In contrast to the cytoplasmic microfibrillae, the chromosomal microfibrillae appear dense, thin and less orderly in their course and arrangement (Figures 4*D* and 5*D*). The lack of orderliness is considered due to the stage. Later when the mature sperm are united into bundles the microfibrillae appear closely packed and orderly—but then they cannot be resolved (cf. Grassé *et al.*[2] last figure of their last plate). The "finest" or least compound ones in Figure 4*D* measure about 30 A and can be followed for a distance of 1000 A and more. Figure 6 illustrates the ultimate "visible" structure of a group of fibrillae taken from the photograph of Figure 4*A* and its through-focus mate (not shown) in the region indicated in Figure 4*A*. The interior of the chromosome is incompletely resolved but at the upper margin three microfibrillae (X, Y and Z in Figure 4*A*) protrude. From this area of the original photograph outlined in Figure 5*A*, Figure 6 was drawn, as the best example of chromosomal microfibrillae obtainable in this series. Thus V, Y, Z in Figure 5*A* correspond to V, Y, Z in Figure 6; X of Figure 5*A* is not shown in Figure 6. In Figure 6 the 30 A chromosomal microfibrilla appears to be a cylindrical structure.

Structural details of the cortices of the microfibrillae are for the most part obscure. However in certain places there is a suggestion of still finer fibrils measuring approximately 15-20 A, e.g. at the end of the structure labelled V and at the lower end of the structure labelled Y (Figure 6). Evidence for the existence of these structures is their appearance in successive negatives differing in focus.

In a series of papers on light microscopy summarized by Nebel[5] it was shown that coiled half chromatids in *Tradescantia* form a pair of transparent helices closely fitted into one another in pairs of compensating coils. Modern authors have suggested a similar structural principle at the molecular level. Figure 6 is not incompatible with such a possibility. Whatever the final answer may be the present figures bring the visible microfibrillae closer to the molecular model. In the absence of biochemical proof other than the Feulgen-positive reaction of the mass of the 30 A chromosomal microfibrillae (Hughes-Schrader[3]), it is assumed that the smallest units may represent the ultimate DNA chains encased in protein of relatively limited mass, possibly protamine.

The structure of the 30 A chromosomal microfibril, as observed in the coccid, approaches in size the postulated molecular dimensions of the DNA core by a factor of 3-6. This unit as I see it consequently appears as the chief component of what is currently called the "multiple" gene string. I have not seen a central master strand or evidence of any transverse "governor" which would make many gene strings behave as one. The number of 30 A chromosomal microfibrillae per chromosome may be calculated easily by dividing the cross-sectional area of the chromosome by the size of the fibrils, i.e. 30 A plus about 200 A interspace. This gives an estimate of between 50 and 100 microfibrils per chromosome. Figure 4*C* confirms such an estimate directly. Here in slightly acid fixation some 60 chromosomal microfibrillae are seen arranged in a V-shaped profile representing, it is assumed, the presence of two optically fused chromatids. This observation implies that a given gene locus probably consists of about 64 identical physical units. Each of the 64 units may be compound, but their ultimate structures are beyond the range of present methods.

Figures 4*B* and 5*B* from the same gonad, also in slightly acid fixation, show the migration of chromatin through the ostium of the tail cap. The microfibrillae here are granular in structure and it is believed that the fixation has caused discontinuities in the strict longitudinal coherence of individual strands. At the upper margin of the ostium two highly multiple strands project into the lumen of the nucleus. In addition there appear to be streams of chromatin following the nuclear wall shown on either side of the ostium. This was implied by Hughes-Schrader[3] (in her figures 4 through 8, plate 1). We know nothing beyond what she has described concerning cytogenetic order within the tail sheath. The apparent lack of order observed in electron micrographs (Figure 4*D* and especially the area from Figure 4*A* which is drawn in Figure 6) may result from the action of the knife used in sectioning and may also in part or predominantly be due to the phase of the spiralization cycle as described by the same author.

Discussion and Summary

The choice of *Steatococcus* sperm for study of chromosome structure is more satisfactory than, e.g., that of *Triturus*, *Tradescantia* or *Lilium*. The reason may be that the microfibrillar components in these chromosomes carry less protein coating on the DNA chain. The chromosome of *Steatococcus* sperm, according to these observations, consists of about 64 microfibrillae each measuring about 30 A in diameter. Each one of these may be compound (cf. Wilkins[8]) containing two or more threads of molecular order. The 30 A chromosomal microfibrils seen in the iceryine coccid chromosome correspond in size to the cores mentioned by Ris[7] and Kaufmann[4], who described heavily coated chromosomal microfibrillae with an invisible central core 40 A

thick. However, the present observations suggest that the iceryine coccid differs in that the protein coating of the DNA core of individual microfibrillae is much thinner, giving a thickness ratio of about 6 (30/5) in the iceryine coccid as compared to about 40 (200/5) in *Triturus* and *Tradescantia*. The difference would appear to lie only in the material used and not in the basic approach or interpretation.

Many current papers dealing with spermogenesis under electron microscopy show microfibrillar chromosome structure in the early stages of the sperm proper. The work of Grassé *et al.*[2] is probably the finest example. As more attention is focused on this stage it may become more than a curiosity. Grassé's term chromonema seems not applicable to any microfibrillae of less than 1000 A diameter (v.i.).

The gap between observation and theory (e.g. the Watson-Crick model) is now narrowed to less than one order of magnitude. Thus it is more than ever necessary to realize that the precise course of the ultimate molecular DNA chains within the microfibrillae and their exact number cannot be described with certainty at the present time.

The term *eltene* (strand) is suggested as the name for the 30 A or larger chromosomal microfibrils which can be resolved only under electron microscopy. As chromonema applies to light microscopy, eltene may describe corresponding structures at the higher resolution of the electron microscope. Phylogenetically the term may be useful to describe the physical equivalent suggested by such concepts as polyteny. Thus a genus X may contain a number of species with the same chromosome number but species A and B may differ with regard to their eltene threads.

Addendum: Dr. Robertis, describes a 28 A chromosomal microfibrilla in *Laplatacris dispar* in *J.B.B.C.* 2:785-796. 1956.

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MUTANT STOCKS OF CATS AND DOGS OFFERED FOR RESEARCH

THE Jackson Memorial Laboratory, Bar Harbor, Maine is offering, for a limited period of time, two types of mutant cats and two types of mutant dogs for use as research material.

Dr. C. C. Little gives a brief description below of the genetic behavior of the cat mutants—the Four-Eared and the Yellow Siamese Cat.

There are also two very important genetic stocks of dogs which will be offered to interested geneticists for breeding. One is a recessive mutant which reduces the size and function of the outside toes on the front and sometimes the hind feet, with the result that the affected animals are really "two-toed" on those feet. To many this is extremely interesting from the evolutionary point of view. The second stock consists of two closely related inbred families of beagles. These show an extraordinary syndrome, involving malocclusion, cleft palate, eye and ear abnormalities, hydrocephaly, and other brain defects, nervous seizures, cryptorchism, and some evidence of other hormonal unbalance. This syndrome is *not* a simple Mendelian

character, but *is* definitely dependent, to a large degree on genetic factors. Anyone interested in using these animals as research material should address his inquiry to the Director of the Laboratory, Dr. Earl L. Green.

FOUR-EARS, A RECESSIVE MUTATION IN THE CAT

C. C. LITTLE*

A TABBY female cat with a small extra pinna on each side, reduced eyes, and slightly undershot jaws was born in Ashtabula, Ohio in 1946. This animal was kindly contributed to the Jackson Laboratory by its owner, Mrs. George Wood.

From this animal, ♀ No. 140, 16 progeny were obtained by normal sires. They were all normal in appearance.

Crossed with her heterozygous son, No. 184, she gave six kittens, 3 normal, 3 "four-eared."

A single litter out of her by a "four-eared" grandson consisted of four animals, all "four-eared."

This evidence that "four-eared" is a simple Mendelian recessive has been strengthened by other matings, as follows:

	Normal	Four-eared
Normal *NN* × *nn* four-eared	38	0
Normal *Nn* × *Nn* normal	20	6
Normal *Nn* × *nn* four-eared	42	30
Four-eared *nn* × *nn* four-eared	0	4

Although there is a deficiency of four-eared animals, the case seems to be well established.

Certain attributes of the four-eared character are interesting.

1. It has *always* been *bilateral*, both as regards ears and eyes. This is unusual in mammalian morphological mutants.

2. It appears to affect the physiological activity and temperament, making the animal less active and more lethargic, although body size seems to be normal.

3. The eyeballs may be greatly reduced and otherwise affected. The neuro-anatomy of the mutant would be of great interest for further study, as would its behavior.

4. The mutant appears to be genetically independent of *A*, tabby, and *sp*, piebald spotting.

THE YELLOW SIAMESE CAT

THE object of this communication is to record the production of yellow cats with the Siamese pattern, and to report briefly their genetic behavior.

The Siamese pattern c^s is a simple Mendelian recessive to full pigmentation, *C*. It has been commonly considered an allele in the *C* series of alleles; the other members being full pigmentation *C*, chinchilla (silver) c^{ch}, and Burmese c^b.

Black and yellow in cats are alternative coat colors. Various symbols have been used in describing them, but since they appear to be chiefly concerned with extension or restriction of black pigment, they will be considered as representing the *E* locus. Black cats will be *E*, yellow *e*.

The *E* locus in cats is on the *X* chromosome, so that black females are *EXEX*, yellow females *eXeX;* black males *EX-* and yellow males *eX-*. Females which are *EXeX* are tortoiseshell.

Tjebbes[1] and others have hypothesized that the black of Siamese cats is a dominant to all other colors, including tabby (agouti *A*), which is epistatic to ordinary black.

Tjebbes cross of a Siamese ♀ × dark tabby ♂ gave 3 F_1 animals, all black. A similar cross recorded by the writer gave a litter of five, all black.

On the other hand, the reciprocal cross observed by the writer, tabby ♀ × Siamese ♂ has given the following result: 5 tabby ♂♂, 3 tabby ♀♀, 2 black ♂♂, 1 black ♀. It appears, therefore, that there are probably Siamese in which the black is dominant to tabby and others in which it is not.

In the crosses made at the Jackson Laboratory, it would appear that the "dominant" black, if present, is an allele of *E*, since no evidence of its independence on a gene in a different locus has been obtained.

Experimental Results

The yellow gene, *e*, was introduced by mating a black female, heterozygous for Siamese, *aaCc*^{s} *EXEX*, with a yellow non-Siamese, *aaCCeX-*. Two tortoiseshell females, No. 7 and No. 8, were obtained. There was an equal chance that these could be *CC* or *Cc*^{s} in formula.

Actually, No. 7, which in appearance showed no Siamese conformation, proved to be *CC*, and No. 8, which in conformation resembled the F_1 Siamese × common tabby turned out to be *Cc*^{s}.

The various matings of No. 7 are as follows:

Tortoiseshell No. 7 ♀ *aaCCEe* × ♂ No.
135 *aac*^{s}*c*^{s}*E-* Black Siamese
3 black ♂♂ : 1 yellow ♂

*Jackson Memorial Laboratory. This study was supported in part by a grant from the Guggenheim Foundation.

Tortoiseshell No. 7 ♀ *aaCCEe* × ♂ No. 12 *aaCCe-* Yellow
6 yellow ♀♀ : 4 yellow ♂♂ : 9 black ♂♂ : 0 black ♀ : 3 tortoiseshell ♀♀

No descendants of No. 7 have given Siamese offspring.

Tortoiseshell females crossed with black Siamese males have given the progeny shown in Table I.

Tortoiseshell females *aaCc^{s}Ee* crossed with yellow Siamese males *aac^{s}c^{s}e-* gave the results shown in Table II.

Results of tortoiseshell Siamese ♀♀ *aac^{s}c^{s}Ee* × yellow Siamese ♂♂ *aac^{s}c^{s}e-* are shown in Table III.

Yellow Siamese × yellow Siamese have, as expected, given only yellow Siamese in over 60 progeny.

Discussion

One of the interesting observations is that yellow cats with or without the Siamese pattern have always shown the "tabby" pattern, whether or not they are genetically *A*. In other words, *aa* yellow cats show either the narrow striped "catus" pattern or the broad striped dark "torquatus" type. Without the *A* gene to bring this out, the blacks and tortoiseshell animals in the same litter do *not* show the tabby striping.

It was, however, observed that in the juvenile stage lightly pigmented Siamese kittens frequently showed a tabby "ghost" pattern, which later disappeared as the pigmentation increased.

The presence of the tabby pattern in yellow or orange cats shows that the pattern is *not* primarily a matter of alternating black and yellow pigment, but of alternation of deep or plentiful pigment formation with decreased pigment formation.

It is probable that the basic striped pattern is present in all cats, not only in all "non-Siamese" as suggested by Tjebbes.

What we have hitherto called "tabby" is, therefore, action of an agouti type of gene in the *A* locus, which is affected in its expression by the basic striping.

The yellow Siamese have clear light blue eyes. The points are yellow or orange and show basic striping which is particularly noticeable in the tail. The body varies from almost pure white to light straw or cream color. This deepens with age, especially in males.

It is interesting that the Siamese pattern in *ee* yellow animals does *not* remove pigment from the points or, at times, even from the coat. This is in contrast with the action of the Himalayan c^h gene in rabbits and guinea pigs where the *ee* forms are pure white. The homology of c^h and c^s is therefore doubtful.

The tortoiseshell Siamese also bear out the difference by having irregular dark or lighter spots on the body. This condition is not seen in Himalayan tortoiseshell guinea pigs.

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TABLE III. ***aac^{s}c^{s}Ee* Siamese tortoiseshell ♀♀ × yellow Siamese ♂♂ *aac^{s}c^{s}e-***

♀ No.	Siamese Tortoiseshell ♀♀	Siamese Black ♂♂	Siamese Yellow ♀	Siamese Yellow ♂♂
A	6	1		3
B	3	2		
F	1	2	1	1
Total	10	5	1	4 (20)
Expected	5	5	5	5 (20)

TABLE I. ***aaCc^{s}Ee* Tortoiseshell ♀♀ × Black Siamese ♂♂ *aac^{s}c^{s}E-***

♀ No.	Black ♀♀	Black ♂♂	Siamese Black ♀♀	Siamese Black ♂♂	Tortoiseshell ♀♀	Siamese-Tortoiseshell ♀♀	Yellow ♂♂	Siamese Yellow ♂♂
8	1	5	7	1	5	1	3	4
17	1	1	1	3	1		2	
42						1		
139		2	4	3	2			
149	4	1	1	2	2		1	2
168	1		2	1		1	2	2
169	1	2		1		2	1	
Total	10	11	15	11	10	5	9	8 (79)
Expected	10	10	10	10	10	10	10	10 (80)

TABLE II. ***aaCc^{s}Ee* Tortoiseshell ♀♀ × Yellow Siamese ♂♂ *aac^{s}c^{s}e-***

♀ No.	Black ♀♀	Black ♂♂	Siamese Black ♀♀	Siamese Black ♂♂	Tortoiseshell ♀♀	Siamese Tortoiseshell ♀♀	Yellow ♂	Siamese Yellow ♂♂	Yellow ♀	Siamese Yellow ♀♀
8	1			2						2
139		6		1	2		5			
139						1		2		
168				1	2		1		1	
169		1								4
Total	1*	7	0	6	4	1	6	2	1	6 (34)
Expected	0	4.5	0	4.5	4.5	4.5	4.5	4.5	4.5	4.5 (36)

*Exception to sex-linked inheritance.

DUPLICATED LINKAGE GROUPS IN COTTON

CLAUDE L. RHYNE*

FOUR linkage groups have been established in allotetraploid Upland Cotton, *Gossypium hirsutum* L. Two of these are used in the present study and contain, according to Stephens[7], on the *A* genome the four linked loci: *R2* (red plant-petal spot), *Yg2* (normal for yellow-green), *Lc1* (brown lint), *N* (fuzzless seed), and *Cl2* (normal for short-branch, linked with *R2* but not yet placed relative to *Yg2*), and on the *D* genome, the two linked loci: *R1* (red plant parts), and *Cl1* (normal for cluster fruiting-habit).

The present study concerns the linkage of *Yg1* for yellow-green and *Dw* for dirty-white lint-color, with the *R1* locus. With the addition of these two loci to the *R1* linkage group, two linkages are shown to have a number of duplicated loci; thus it is suggested that two duplicate linkage groups are present in the allotetraploid cotton species.

Several duplicate loci have been reported in cotton. Most of these may be considered to be duplicated in, or have been doubtfully linked in, the two linkage groups used in the present study. For example, Harland[2] considered *R1* and *R2* to be duplicates, each occurring in a different genome and each influencing anthocyanin production in various plant tissues. Harland also considers his K^b (which may be the same as *Dw*) and K^h (= *Lc1*) loci to be duplicates, each producing brown-linted cotton and each doubtfully linked with one of the anthocyanin loci. Likewise, Rhyne[5] established that duplicate loci are involved in yellow-green inheritance, either *Yg1* or *Yg2* producing a normal plant phenotype. Rhyne[5] considered that *Yg2* and *Lc1* were linked with *R2* of the *A* genome and suggested that *Yg1* and *Dw* were linked with *R1* of the *D* genome of Upland cotton. Stephens[7] has confirmed *Yg2* and *Lc1* linkage with *R2* therefore a proof of linkage for *Yg1* and *Dw* with the *D* genome *R1* locus would suggest a duplicated linkage group. Data establishing linkage in support of this hypothesis of duplicated linkage groups in cotton are to be presented and discussed herein.

Establishing *R1-Yg1* Linkage

Representatives of three allotetraploid *Gossypium* species were known to have the *Yg2* and *Yg1* duplicate genes[5]. A line of each allotetraploid species was hybridized with a genetic tester stock of Upland cotton (*G. hirsutum*) which had the *A* genome-linked genes *r2yg2* and the assumed *D* genome linkage *R1-yg1*. The F_1 hybrids may be written: *A* genomes $\frac{R2\text{-}Yg2}{r2\text{-}yg2}$ in coupling linkage and *D* genomes $\frac{r1\text{-}Yg1}{R1\text{-}yg1}$ in repulsion linkage. These F_1 hybrids were backcrossed reciprocally with a recessive stock that had the *r2-yg2* and *r1-yg1* genes. The chosen lines of the parental allotetraploid species exhibited similar anthocyanin expressions on the plant parts caused by their dominant *R2* genes.

The data on the scoring of the backcrosses are shown in Table I. Either *R1* or *R2* dominant produced a red plant-color phenotype and only *r1r1r2r2* produced a non-red phenotype. Similarly, either *Yg1* or *Yg2* dominant produced a normal plant but only a *yg1yg1-yg2yg2* genotype produced yellow-green plants. The expectation for the back-

*Research Asst. Prof. and Geneticist, F.C.R.B. Joint contribution from the Field Crops Department, North Carolina Agricultural Experiment Station, Raleigh, N. C. in cooperation with the F.C.R.B., A.R.S., U.S.D.A. Published with the approval of the Director of Research as Paper No. 751 of the Journal Series.

TABLE I. Backcross segregation of anthocyanin and yellow-green from three interspecific hybrids*

F1 Hybrids used*		Phenotypic Segregation			
		Red Plant-Color		Non-Red Plant-Color	
		Normal	Yellow-Green	Normal	Yellow-Green
G. hirsutum L. × *G. barbadense* (T169 × Red-yellow)	Observed Numbers	91	46	41	5
	Expected if *r1–Yg1* Independ.†	(109.8) = 60%	(27.5) = 15%	(27.4) = 15%	(18.3) = 10%
	Expected if *r1–Yg1* linked‡	(98.8) = 54%	(38.4) = 21%	(38.4) = 21%	(7.3) = 4%
G. hirsutum × *G. hirsutum* (MZ106 × Red-yellow)	Observed Numbers	38	21	19	2
	Expected if *r1–Yg1* Independ.*	(48.0) = 60%	(12.0) = 15%	(12.0) = 15%	(8.0) = 10%
	Expected if *r1–Yg1* linked†	(43.2) = 54%	(16.8) = 21%	(16.8) = 21%	(3.2) = 4%
Extract from *G. arboreum–G. thurberi* × *G. hirsutum*	Observed Numbers	26	11	16	0
	Expected if *r1–Yg1* Independ.*	(31.8) = 60%	(7.9) = 15%	(7.9) = 15%	(5.3) = 10%
	Expected if *r1–Yg1* linked†	(28.6) = 54%	(11.1) = 21%	(11.1) = 21%	(2.0) = 4%

*F1 $\frac{R2\text{–}Yg2}{r2\text{–}g2}$ $\frac{r1\text{–}Yg1}{R1\text{–}g1}$ backcrossed to $\frac{r2yg2}{r2yg2}$ $\frac{r1yg1}{r1\text{–}g1}$. Either *R1* or *R2* produce reddish plants, and either *Yg1* or *Yg2* produce normal plants.

†12: 3: 3: 2 ratio using the *R2–Yg2* linkage value of 20 percent after Stephens[7].

‡This assumes 20 percent recombination values between *R2–Yg2* and *R1–yg1*.

crosses of Table I, assuming a 20 percent linkage at *R2-Yg2*[7] and independence for *r1* and *Yg1*, was a ratio of 12:3:3:2. The observed numbers in each phenotype are different from the expected numbers. An excess in the parental classes, the second and third phenotypes of Table I, is suggestive of linkage between *r1* and *Yg1*. Upon referring to Table II where the chi square analysis shows a significant interaction, it is apparent that *r1* and *Yg1* are not independently inherited.

The alternative hypothesis was that linkage occurs at *r1-Yg1*. The expected ratio, based on 20 percent linkage between both duplicate *R* and *Yg* loci, is given as the third row of each hybrid in Table I. The observed number in each phenotype and the expected number are similar in magnitude. Again upon referring to Table II where the interaction is nonsignificant, the writer accepts a hypothesis that *r1* and *Yg1* are linked, as has been shown for *R2* and *Yg2*[5, 7]. The data from the backcrosses of F_1 hybrids are interpreted as *R* and *Yg* linkage on each genome.

Establishing *R1-Dw* Linkage

A necessary line was produced that had a *Dw* allele from parental *G. raimondii* Ulb. transferred into a Z106 double-haploid stock of *G. hirsutum*. The gametic composition was *Rai-Dw*, where *Rai* represents the allele of *G. raimondii* at the *R1* locus[1, 4] on the *D* genome. The stock when again crossed with Z106 gave the F_1 $\frac{Rai\text{-}Dw}{r1\text{-}dw}$. An F_2 was grown and classified. Table III contains 244 plants scored in a study from a larger population. Both *Dw* and *Rai* were scored in the population; however, only plants multiply checked for anthocyanin expression were used.

The classifications in Table III are presented in four phenotypes for simplicity, although the incompletely dominant *Rai* and *Dw* genes were scored in nine phenotypes. Classification was done by using the method of Gerstel[1] and Rhyne[4] where *Rai* is indicated by red-

dish and *r1r1* by colorless anther filaments. *Dw* produced a dirty-white seed fuzz while *dwdw* produced normal white fuzz and white lint. The observed number in each phenotype differs from that expected from a hypothesis of independent inheritance. Using the chi-square analysis shown at the bottom of Table III, the signfiicant interaction is interpreted to indicate *Rai (R1)-Dw* linkage. A value of 27.45 percent was calculated for the linkage when Murty's[8] method of minimum discrepancy was used.

Either *Dw* or *Lc1* (of the *R2-Yg2-Lc1* linkage) produces brown lint. As each shows linkage with one of the anthocyanin duplicates, duplicate *R-Lc* linkages may be considered to be present on each genome of Upland cotton. The linkage of ca. 27 percent of *R1-Dw* cannot be considered a duplicate of the 50 percent linkage between *R2-Lc1* (see Stephens[7]). The estimate, however, could be indicative of a chromosome structural difference. After all the *R2* chromosomes of the *A* genome and the *R1* chromosomes of the *D* genome do not form quadrivalents, they show normal bivalent pairing. Therefore, one would expect differences as well as similarities, and it should not be surprising to find a significant difference in linkage values between two duplicated loci. Additional studies are in progress on the nature of the reduction of the *R1-Dw* linkage.

Discussion

The linkage group containing *R1-cl1*[7] has been extended to include the *R1 (cl1)-Yg1-Dw* loci. All of its loci have been shown to be located on the *D* genome[1, 2, 4, 5, 7]. Stephens[7] extended the *R2-cl2* linkage to contain *R2-cl2-Yg2-Lc1-N*, and all loci have been shown to be on the *A* genome[2, 4, 5, 7]. The data thus support Rhyne[5] that two multiple-locus, linkage groups are present in duplicate in the allotetraploid cotton species.

The actual value for each *A* genome *R2-Yg2* and every *D* genome *r1-Yg1* linkage is not necessarily the 20 percent found to give the best fit in Table II. Rather, devi-

TABLE II. Chi-square analysis of major gene segregation in hybrids from Table I.

F_1 Hybrid		Segregation Expected		Interaction	
		Anthocyanin *Rl* vs *rl* 1 : 1	Yellow-green *Yg* vs *yg2yg2 yg1yg1* 3 : 1	Independent *r1–Yg1** inheritance	Linkage of 20% for *r1–Yg1**
T169 × *Ryg*	Chi-square	2.890	0.803	36.214	1.339
	P for Idf	0.10-0.05	0.50-0.30	0.01-0.001	0.30-0.20
MZ106 × *Ryg*	Chi-square	0.050	0.600	22.816	2.607
	P for Idf	0.80-0.70	0.50-0.30	0.01-0.001	0.20-0.10
Dl rCl × *Ryg*	Chi-square	0.924	0.509	16.359	3.774
	P for Idf	0.50-0.30	0.50-0.30	0.01-0.001	0.10-0.05

*Assuming the known linkage *R2–Yg2* is 20 percent after Stephens[7].

TABLE III. Anthocyanin and lint-color segregation in F_2 progeny. Coupling phase on genotype (Z106) of *G. hirsutum*

F_1 hybrid Genotype	Phenotypic Segregation			
	Anther Filaments Red		Anther Filaments Clear	
rl-dw / *Rai-Dw*	Dirty-White Seed Fuzz *Rai- Dw-*	White Lint Not Dirty-White Fuzz *Rai- dwdw*	Dirty-White Seed Fuzz *rl rl Dw-*	White Lint Not Dirty-White Fuzz *rlrl dwdw*
Observed Numbers	162	24	34	24
Expected, if Independent	(137.25) 9/16	(45.75) 3/16	(45.75) 3/16	(15.25) 1/16
Chi-square Analysis	*Rai* vs *rl*	Idf	0.088	P= 0.80-0.70
	Dw vs *dw*	Idf	3.090	P = 0.10-0.05
	Linkage	Idf	19.288	P = 0.01-0.001

Recombination calculated at 24.75 percent after Murty[8]

ation from a standard value, say 20 percent for *G. hirsutum* linkage at *R-Yg*, can be considered when found in a related genome, as an indication of genic, cryptic difference between the two related species[6]. That is, the *R-Yg* linkages may be useful for evaluating cryptic structural difference among *Gossypium* species having a common genome.

Multiple locus, duplicated linkage groups were suggested by Rhyne[5] for use in studies relating to a low metaphase chiasmata frequency that is observed in cotton. Three-point linkage studies were suggested specifically using the *R-cl-yg* loci available within Upland cotton.* Presumably all loci are located in a distal end of each linkage group. Even so, *cl1* and *cl2* have not been placed relative to *Yg1* and *Yg2* respectively, although the total genetic map distance could be as low as 20 percent (order *R-cl-yg*) or as high as 38-40 percent (order *cl-Ryg*). Therefore, according to Stephen's[7] hypothesis, a single chiasma would be formed in a chromosome arm, as cytological observations at metaphase show, and thus double recombination would not be expected from genetic three-point linkage tests. Alternatively double recombination, when occurring, could suggest that chiasmata frequencies are greater than the frequency observed at metaphase.

Complications, however, have been encountered when attempting three-point tests among the linked loci of either duplicated linkage group. For examples: (A) When testing *D* genome *R1cl1, Yg1 and Yg2* were present in duplicate. Apparent *R1-cl1-Yg1* recombination was shown to be parental *R1-cl1-yg1* + *Yg2* after genetic testing. Table I, for instance, has been complicated by duplicate *Yg2* segregation, and the *r1-Yg1* linkage value of 20 percent reported there is an average best fit *when* both linkages are present. Backcrossing is necessary to obtain a *r1-Cl1-yg1* phenotype without the complicating *Yg2* genes. The *Cl1* and *Cl2* loci can be expected to give similar complications in some interspecific hybrids. (B) When testing *r1-yg1-dw, Yg1* and *Yg2* were present. Both *r1-Yg1* and *r1-dw* linkages were found, confirming data shown in Table I and Table III, but *yg1-dw1* linkage was poorly estimated. Apparently, *yg1-dw* linkage is tight, but is not 0.0 as the data suggested when all recovered *yg1yg1* (*yg2yg2*)-*dwdw* plants were of the combination found in one parent. No estimate of *r1-cl1-dw* linkage was attempted since available stocks had green fuzz (at locus *Cr*, see[7]) which interferes with *Dw* expression. (C) When *R2-Cl2-Yg2* was transferred from the A_2 genome (*G. arboreum* L.) to Upland cotton, *Cl1* and *Yg1* were both present (using technique for the third hybrid of Table I). Both *Cl1* and *Yg1* were removed after additional backcrossing on the only available *yg2 R1-cl1-yg1* stock. Although *C12* was dominant over *cl1-cl1, R2* could not be classed with accuracy when *R1R1* was present. (D) A tester has not been produced, perhaps because of insufficient numbers, that has *D* genome *r1-cl1-yg1-dw* and *A* genome *r2-yg2-cl2-n*. The *D* genome genetic composition is common but then *Yg2* is present. Substitution of *R1 for r1* on the *D* genome plus the elimination of *Yg2* has been completed. This stock is subject to the complications in (C) above.

*Current three-point linkage data obtained too late for this publication indicate the order is *Cl1-R1-yg1. Dw*, however, cannot be scored until the plants are mature. No double recombination was detected.

Summary

The *Yg1* duplicate allele for yellow-green was found to be linked with the *R1* locus of the *D* genome of allotetraploid *G. hirsutum*. The estimate was 20 percent, as was the estimate given for *R2-Yg2* linkage on the *A* genomes by Stephens[7]. The *Dw* locus was calculated to show linkage with *R1* of ca. 27 percent. These loci extended the *D* genome linkage group to four loci, *R1-cl1-yg1-dw*, the "duplicates" of those reported for the *A* genome linkage group, *R2-cl2-yg2-Lc1-n*[7]. The gene order is not necessarily duplicated in both linkage groups. Either of the linkage groups may be useful in three-point linkage tests and the genetic data should be related to the question of chiasmata frequency and position within *Gossypium*.

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SCALELESS, AN INHERITED ECTODERMAL DEFECT IN THE DOMESTIC FOWL

U. K. Abbott and V. S. Asmundson*

ABNORMALITIES of the plumage of the domestic fowl have been known for a long time. At least seven distinct mutations which produce structural changes in the plumage without affecting pterylosis have been identified. These include: frizzle[14,21], silkie[1], flightless[29], fray[30], naked[18], sunsuit[16] which resembles stringy[2], and woolly[19]. Birds with all of these mutations have an apparently normal skin structure although other ectodermal derivatives may be affected as in the case of sunsuit (stringy), where overgrowth of the beak and nails has suggested a dyskeratosis as the cause of all the epidermal changes. Alterations in feathers but not in pterylosis or skin structure have also been found in other bird species including the "penguin" guinea fowl[22], the "porcupine" pigeon[3,20], the naked pigeon[5], the silky pigeon[4] and the silky dove[23].

Two other mutations affecting the plumage of the domestic fowl interfere with pterylosis and consequently restrict feathering to certain areas of the body. In naked-neck, a viable dominant differentiating a breed of fowl[7,10], the pterylae in the head, neck and ventral regions of the body are absent or much reduced in area and the apteria lack feathers entirely. In autosomal apterylosis[25,26] the reduction in feather tracts is even more extreme. The anterior and posterior spinal, the humeral and the femoral tracts are most restricted. The crural tract is slightly reduced while the head and alar tracts may be unaffected.

Inherited abnormalities of the epidermal covering are also known in most other animal species. In general, these, like the majority reported in the fowl affect the emerging hair, the regenerating follicle or the structure of the hair rather than the distribution of follicles. In cattle inherited defects of the coat range from the semi-hairless condition of Craft and Blizzard[6] to the extreme effects in the case reported by Hadley and Cole[12] and later discussed by several others[5,28,31]. In this instance hairlessness is a consequence of other underlying ectodermal abnormalities. In between these extremes is viable genetic hypotrichosis[17,27] in which the hair is absent from most of the body and the follicles are abnormal.

Grüneberg[11] has recently reviewed the mutations in the house mouse affecting hair distribution, pattern and growth. In hairless and rhino, a hyperplasia and hyperkeratosis of the stratified epithelium are blamed for the failure of hair to regenerate normally following loss of a normal baby coat 10-14 days after parturition. In these mutations the entire hair falls out but in naked, another mutant of the mouse causing a loss of hair shortly after birth, the hairs break off at the skin surface in many regions of the body apparently because of an incomplete keratinization, also indicated in homozygotes by an excessively thin skin.

Origin of Scaleless

A new mutation which we have called scaleless was found among New Hampshire chicks from the University of California flock at Davis in the autumn of 1954. At that time one male and four females carrying the new mutation were identified. All were sired by male no. 2002, the four carrier females representing approximately half of his tested progeny. The dams of the carrier hens were not closely related to the carrier male nor to each other, suggesting that male no. 2002 was the original carrier.

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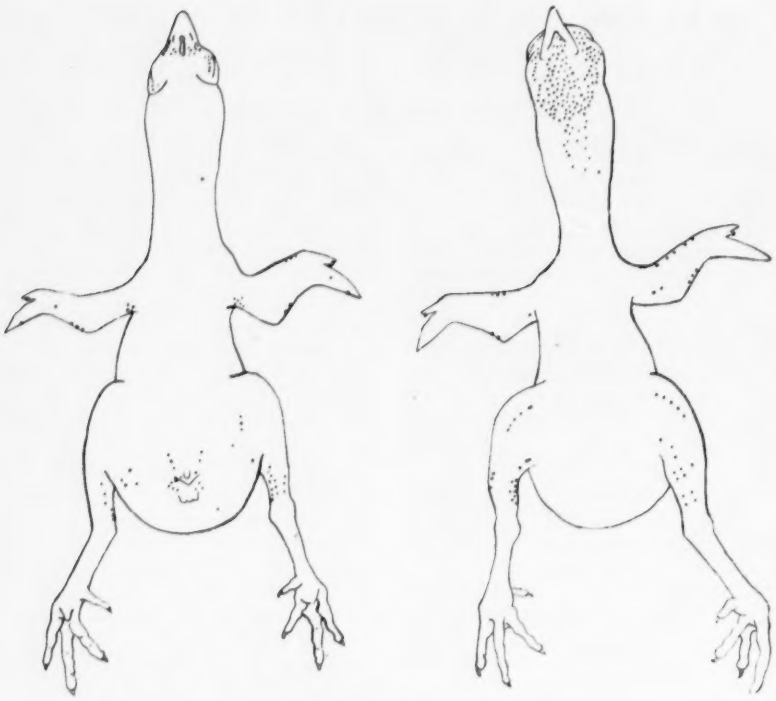


DISTRIBUTION OF FOLLICLES

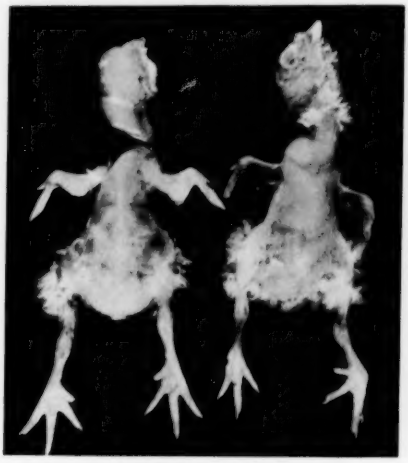
Figure 7

The distribution of feather follicles on a typical day-old scaleless chick is illustrated, showing dorsal and ventral views.

Morphology of Scaleless

A. *In the chick*

Although the most noticeable characteristic of scaleless chicks is the nearly complete absence of down feathers, the name scaleless has been chosen specifically to differentiate this type of nakedness from others known in birds. For the defect in this instance occurs earlier in embryogeny and prevents the appearance of all scales as well as most of the specialized derivatives of scales, which include feathers, foot pads and spurs. The skin not only lacks feathers in all but a few specific areas, but also lacks the majority of feather follicles normally present in specific tracts or pterylae. The few down feathers present occur principally in the head tract, with less in the femoral, crural and caudal tracts. The major feather tracts are completely bare of feathers. Figure 7 illustrates the distribution of follicles in a typical day-old scaleless chick. Figure 8 shows a day-old scaleless chick with down intact. The epidermis of these chicks (except in the location of the few follicles) is waxy and smooth and transparent over the body proper. The skin of the legs, while somewhat thicker than that of the body, is completely devoid of scales and is smooth in texture. The under surface of the foot which in normal



SCALELESS CHICK
Figure 8

A day-old scaleless chick with down intact —dorsal and ventral views.

chicks is covered with specialized scale derivatives (organized into foot pads which provide the birds with traction) is also smooth, although the skin is folded in the manner typical of foot pads (Figure 9*A*). Day-old scaleless chicks do not differ in weight from normal chicks from New Hampshire eggs of comparable size and strain.

B. *In the adult*

During the growing period when the primary down feathering is successively replaced by several changes of juvenile feathers and finally by adult feathers, scaleless birds become even more naked. Areas which had a scant number of down feathers at hatching time are often bare at maturity. This is particularly true of the head tract (Figure 10) where a number of follicles appear incapable of function after loss of the original down feathers. Adult feathers are small and abnormal in structure; they are primitive, resembling the least specialized juvenile rather than adult, plumage. Feathers from different areas of the body are structurally very similar, not highly differentially specialized as is the case in normal fowl. During the normal moulting period scaleless females cease egg production and replace their few feathers. The female illustrated in Figure 10 has just completed her moult and has, accordingly, the maximum number of feathers present in her adult life. A few weeks before her moult she had even fewer feathers. The head tract remains relatively bare after moulting. The appearance of new feathers after moulting indicates that the few follicles present in the juvenile and adult retain their function throughout life.

The bare skin on scaleless birds, particularly on the head, neck and in the region of the vent becomes reddish at maturity. Spurs fail to develop on males. Other ectodermal derivatives such as nails, beak, comb and wattles are apparently normal. Figure 9*B* presents a view of the epidermis of a normal and a scaleless leg belonging to the same adult female, an isolated instance of the unilateral expression of the mutation.

Genetics of Scaleless

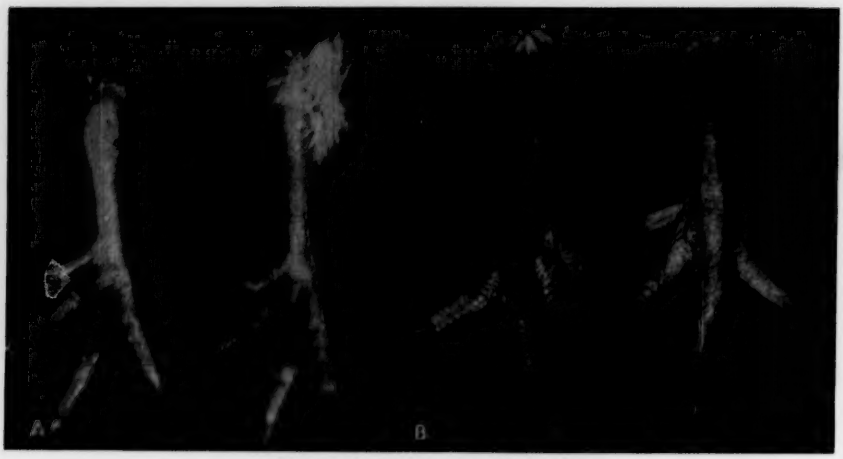
The three types of matings made to study the heredity of scaleless are summarized in Table I. No significant deviations from a 1:1 sex ratio were found in either normal or scaleless progeny. These data indicate that the scaleless phenotype appears in birds homozygous for an autosomal recessive allele, for which the symbol *sc* has been chosen.

Irregularities in the Inheritance of Scaleless

One mating of scaleless carriers produced a plumage mosaic which like Goodwin's mosaic[9] was otherwise grossly normal. Previously the mating in ques-

TABLE I. Proportions of normal, scaleless and unidentified (early dead) progeny

Year	Parents		Number				χ^2
	Sire	Dam	Fertile	normal	Scaleless	Unidentified	
1954	+*sc*	+*sc*	57	37	19	1	2.18 (3N:1 *scsc*)
1955	*scsc*	+*sc*	44	19	22	3	0.22 (1N:1 *scsc*)
1955	*scsc*	*scsc*	147	0	97	50	0.00 (all *scsc*)



SCALELESS AND NORMAL LIMBS

Figure 9

A—Right leg of a scaleless and normal day-old chick. Note skin texture and absence of scales and foot pads. *B*—Right and left limbs from a scaleless mosaic adult female. Note absence of scales on left leg.

tion had given only normal or typical scaleless progeny in a 3:1 ratio. The mosaic, a female (legs illustrated in Figure 9*B*) reached maturity but did not produce any eggs. Autopsy of this bird after its death revealed that it had a small regressed ovary and oviduct with coagulated egg material present and a soft shelled egg in the body cavity. The adrenals and kidneys were enlarged. This bird was normally feathered on the right side of the body and had normal scales and foot pads on the right leg. On the left side she exhibited the typical scaleless syndrome.

In a mating of a normal carrier male with a scaleless female a chick was produced with normal scales and down feathering except for a large featherless patch on the lateral feather tract. In this area the skin had the same characteristics as in typical scaleless chicks. Two other chicks with featherless patches were discovered in New Hampshire chicks of the same strain but in this case the chicks were not from pedigreed stock and it was not possbile to ascertain their relationship to the scaleless line. A further study of this condition is underway to determine whether it is heritable or due to some other cause such as somatic misdivision.

Survival and Performance of Scaleless Birds

Embryonic and Post-Embryonic Viability

No differences were found between fertility or hatchability of fertile eggs from carrier and non-carrier dams mated to the first carrier male identified (Table II), which suggests that the *scsc* genotype is viable during embryonic life. However matings of the more inbred scaleless birds and of normal males to inbred scaleless females produced eggs with relatively poor hatchability (Table III). Matings of scaleless males with carrier females produced equal proportions of normal and scaleless progeny and in this case embryo mortality of both phenotypes was approximately equal. A higher percentage of embryos from crosses of scaleless males with White Leghorn and Barred Plymouth Rock females hatched than from crosses of scaleless females with Cornish and Jungle Fowl males. It is probable that the decreased embryo viability found in third generation embryos reflects inbreeding degeneration resulting from matings of relatives during the process of establishing the scaleless line.

Scaleless chicks were reared in battery brooders in a room maintained between 70°F and 80°F. They grew satisfactorily at ordinary brooding temperatures, about 90° gradually decreased to 80°F., for the first six weeks after hatching. Their growth during this period and to maturity was comparable to that of normal siblings. They did not survive well

under floor brooding in competition with normal chicks. To date, 65 scaleless birds have been successfully reared indoors under controlled temperature conditions. Attempts to raise scaleless birds in outdoor pens (subject to normal diurnal temperature variations) have not been successful save in one case where artificial clothing was provided.

Scaleless females attained sexual maturity between 160 and 180 days at similar ages as normally feathered New Hampshires of the same strain. Their rate of egg production was low (about 40 percent) for birds in full production. Eggs produced by the first generation scaleless females appeared to be more variable in size, shape and shell quality than those from normal hens. It seems possible that metabolism in scaleless females may be altered enough to interfere with normal egg formation. The lower hatching power of fertile eggs from scaleless females (Table III) may also reflect variable shell quality. Further study of this point is necessary however and will be carried out in later, and less inbred, generations.

Differential Resistance to Cold Shocks

Exposed to a 55°F temperature in a refrigerated room immediately after hatching, scaleless chicks died in less than two hours (range, 70-90 minutes) while Siegel and Mueller[24] found that 43.9 percent of 57 normal New Hampshire chicks survived 2½ hours exposed to the more extreme temperature of 37°F.

Adult scaleless males maintained in wire cages with 14 hours of light daily, died after three to five days continuous exposure to temperatures ranging between 53°F and 65°F (mean 58°F) although two survived until after the temperature was raised over 70°F. Females proved more resistant to these conditions; the majority survived the test period (Table IV). Following this period of test the surviving adult females and an additional 11 females moved directly from the indoor controlled temperature room, have been maintained successfully in a room with temperatures ranging between 60°F and 70°F.

Development of Feather Follicles

Normal and scaleless siblings were examined alive at 24 hour intervals from the beginning of the ninth day of incubation until the beginning of the 14th day, or during a period encompassing the first appearance of feather follicles and scales and the subsequent differentiation and growth of feathers. The stages examined correspond to stages 30, 31, 33, 35, 36—40 cited in[13]. The different feather tracts as well as the follicles appear in a specific sequential order corresponding[13] to their destined location and role. In normal embryos, the first papillae appear in rows on either side of the spinal cord at stage 30 which was early on the 9th day for our series in a commercial type incubator. In scaleless embryos the first papillae were detected early on the 11th day of incubation which corresponds to stage 35. Scaleless embryos of this stage had papillae on the head, crural, femoral and caudal areas, i.e. in all areas where feathers occur in scaleless birds. Down feathers were observed in these areas by stage 38. Thus the formation of feather papillae was delayed in scaleless embryos, compared to normals, but in large part this delay reflected the absence of the tracts normally appearing first. Apart from the absence of scales and the tardy appearance of the feather papillae, scaleless embryos were similar to normal embryos in rate of embryonic development. No differences in the body size, or degree of differentiation in other organs such as beak, egg tooth, extent of eyelid closure or limb development were found between normal and scaleless progeny. Histological preparations of skin demonstrated the absence of follicles from most of the body.

Discussion

The scaleless phenotype results from the action of an autosomal recessive gene with full penetrance. The majority of feather mutants which have been identified in chickens as well as other bird species alter either the structure of the feather, its emergence from the follicle or its tenacity rather than the presence or dis-

TABLE II. A comparison of fertility and hatchability of eggs from ++ and +*sc* females mated with +*sc* male, Autumn, 1954

Dam	Dam's genotype	No. eggs set	% Fertile	% Hatch (of fertiles)
H 2	++	20	95.0	100.0
H 8	++	17	100.0	100.0
H 20	++	7	85.7	66.7
H 70	++	12	100.0	75.0
H 93	++	10	100.0	80.0
H106	++	14	92.9	84.6
H121	++	17	100.0	94.1
H177	++	4	100.0	100.0
H179	++	2	50.0	100.0
H194	++	17	100.0	100.0
Total	++	120	96.7	91.4
H 19	+*sc*	17	82.4	85.7
H 47	+*sc*	20	100.0	95.0
H 60	+*sc*	6	100.0	100.0
H236	+*sc*	17	100.0	88.2
Total	+*sc*	60	95.0	91.2

A TYPICAL SCALELESS BIRD

Figure 10

Two views of a typical 18-month-old scaleless female just following completion of moult. Maximum number of feathers are present. Note thin waxy skin, absence of leg scales and appearance of head and neck.

tribution of follicles. Exceptions to this generalization are the dominant allele, naked-neck, and the recessive autosomal apterylosis. Scaleless quite obviously belongs to the same general group of mutants as these latter two but is characterized by a more generalized interference with normal follicle formation than either. Scales as well as all their specialized derivatives including feather papillae, foot pads and spurs are affected.

Scales are differentiated by the action of localized concentrations of mesenchyme in the dermis; these lead to responsive thickenings and outgrowths of the overlying epidermis as flattened papillae[33]. Feather primordia arise in a definite sequence in definite tracts with tract specific rates of growth, first as papillae which later sink back into the epidermis forming pits or follicles. In scaleless the co-ordinant effect on scales, and all scale derivatives suggests that the defect may lie in the organizing mesenchyme. For the appearance of any feather papillae at all indicates that the epidermis itself is competent to form scale deriva-

TABLE III. Hatchability of fertile eggs from matings of scaleless and scaleless carrier birds

Year	Parents		Number fertile eggs	Percent hatch
	Sire	Dam		
1954	+*sc*	+*sc*	57	91.2
1955	*sc*/*sc*	+*sc*	44	68.6
1955	*sc*/*sc*	*sc*/*sc*	147	43.6
1956	*sc*/*sc*	*sc*/*sc*	132	13.8
1956	*sc*/*sc*	++*SCWL**	94	90.4
1956	*sc*/*sc*	++*BPR**	85	84.7
1956	++Cornish*	*sc*/*sc*	63	52.4
1956	++J. F.*	*sc*/*sc*	71	63.4

*In 1956 scaleless New Hampshires were crossed to four different breeds of chickens; White Leghorns, Barred Plymouth Rocks, Cornish and Jungle Fowl.

tives. It is difficult to imagine the type of gradient in ectodermal competence that could account for the observed follicle pattern. This point however, requires further study, possibly by transplantation techniques.

The feather papillae actually formed in scaleless embryos appear in the normally late differentiated tracts well after the developmental stage when the first papillae are to be found in normal embryos. Furthermore they appear in all their eventual locations at once rather than sequentially. Scaleless mutants form only one type of feather in adult life, irrespective of body location. This observation taken together with the uniformly delayed appearance of embryonic feather papillae suggests that we may be dealing with a case like that of the crinkled mouse[8]. A study of embryological stages in crinkled revealed that the characteristic coat texture is due to the presence of only one type of hair and that the two kinds of hair absent from the crinkled coat reflect the suppression of follicle formation in two distinct periods, 12½-17 days of embryonic age and from birth onwards when the follicles of these hairs are normally laid down. The crinkled case has also led to the suggestion that in normal hair formation in the mouse each type of hair arises from its characteristic type of follicle and that the various types of follicles appear in a definite time sequence. We are tempted to suggest that *scsc* is tissue specific and also time specific and that its characteristic action reflects an interference with the type of epidermal cells during the period when scales and scale derivatives are normally differentiated. A failure of mesenchymal cells to evoke an organizing response in the overlying ectoderm during the critical period when such a response is possible would explain the syndrome best.

The scaleless phenotype is fully viable during embryonic life and, under natural conditions for a domestic flock of poultry, completely lethal during post-embryonic life. However, the provision of a suitable environment renders the phenotype almost completely viable during post-embryonic life as well. This fortuitous combination renders it a useful genetic and physiological tool.

On a more speculative basis one might suggest scaleless as a candidate for evolution's class of hopeful monsters. The occurrence of a mutation such as scaleless in a small population of birds located in a warm climate with little diurnal temperature variation (at least during the rearing season) and in the absence of strong predation could serve as the foundation for a distinct new type capable of survival under natural conditions. The accumulation of appropriate modifiers might eventually extend the viable temperature range. It is notable that adult scaleless females with some protection are viable under the "natural" conditions of Davis at present, although males are not.

TABLE IV. Incidence of death among adult scaleless males and females exposed to a mean temperature of 58°F (range 53°F-65°F) for five days

	♂♂	♀♀
No. tested	10	14*
No. dying durnig test period	8	3*
No. dying within two weeks of test	2	1

*1 of the females had a male-like appearance and produced no eggs.

Summary

Scaleless, a new autosomal recessive mutation has been described. The name has been chosen to illustrate its effect on all scales and on the specialized derivatives of scales, feather papillae, spurs and foot pads. Chicks homozygous for *sc* have a smooth, waxy skin, largely free of down feathers; their legs and feet lack scales and foot pads. Other epidermal derivatives such as nails, beak, comb and wattles are normal. A few scattered feathers are present in the head, humeral, crural and caudal tracts. The adult scaleless bird has even fewer feathers than the chick and these are abnormal, resembling juvenile rather than adult plumage. Adult males do not develop spurs.

Feather follicle formation is retarded in scaleless embryos. During the period when the primordia of the scales and major feather tracts characteristically appear, no papillae are evident in *scsc* embryos. Later scattered papillae appear almost simultaneously on the head, humeral, crural and caudal areas.

The scaleless mutation does not affect embryo viability for *scsc* embryos from normal carrier dams hatched as well as their normal siblings. After hatching the *scsc* phenotype is lethal under natural rearing conditions but is viable when birds are reared in rooms maintained at suitable temperatures. Under the latter conditions their growth rate is equal to that of normal birds of the same strain and they attain sexual maturity at the same time. Inbred scaleless females lay at a low rate of 40 per cent, the eggs varying in size, shape and shell quality; relatively fewer embryos hatch from such dams than from normal dams.

Adult scaleless females are more resistant to temperature extremes and to prolonged low temperature exposure than adult males.

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INHERITANCE OF SEEDLING COTYLEDON POSITION IN PHASEOLUS SPECIES

J. Robert Wall and T. L. York*

SEEDLINGS of *Phaseolus* have two distinct cotyledon positions. In *P. coccineus* the cotyledons are hypogaeous while in *P. vulgaris* they are epigaeous. The position of cotyledons in the seedling stage has long been considered a valid taxonomic character in this genus. However, Freytag[1] reports races of *P. coccineus*, found in Mexico in the states of Puebla, Oaxaca, and Chiapas, which bear their cotyledons half-way between the first leaves and ground level, i.e., epigaeously. He also states that in this same region are found races of *P. vulgaris* which have their cotyledons practically touching the ground. This report leads to the inference that interspecific hybridization has occurred between these species.

Lamprecht[2,3,5] has made extensive studies of the cross *P. vulgaris* × *P. coccineus*. He states[5] that the position of the cotyledons on the stem is determined by two alleles of one interspecific gene. His designation for the *vulgaris* allele is *Epi* (for epigean cotyledon) and for the *coccineus* allele is *Hyp* (for hypogean cotyledon). He further states[4] that the *coccineus* allele *Hyp* in *vulgaris* cytoplasm mutates at a rather high frequency to the *vulgaris* allele *Epi*.

The findings to be reported here are not compatible with a mono-factorial interpretation and suggest a complex mode of inheritance.

Materials and Methods

Three pure lines of *P. vulgaris* were used as maternal parents. These were a White Marrow type, 7-16; a selection from Black Turtle Soup (BTS) variety; and a breeding line known as N320-1. In the presentation of results these three varieties are treated as one population. It should be borne in mind, however, that of 81 plants listed for P_1, 32 are BTS, 26 are N320-1, and 23 are 7-16. The one variety of *P. coccineus* used was Scarlet Runner, obtained from the Burpee Seed Company.

Species hybridization was attempted in only one direction since other workers have been unsuccessful in using *P. coccineus* as the female parent in crosses with *P. vulgaris*[5]. The initial interspecific cross was performed in the greenhouse and the F_1, F_2, and F_3 plant generations, except for the seedlings studied in this experiment on cotyledon position, were raised to maturity in the field. *P. vulgaris* is known to be highly self-pollinated and *P. coccineus* highly cross-pollinated. All field-grown plants were without protection from pollen vectors, and it is known that considerable cross-pollination was occurring in each filial generation. The numbers in the field populations were as follows: F_1, 270 plants; F_2, 369 plants; and F_3, 550 plants. The 369 F_2 plants were selected from the F_1 so as to represent the full fertility range of the F_1 plants with the more fertile F_1 plants receiving a proportionally larger representation in F_1. The F_3 population was made up similarly.

It was essential to devise an objective means for precise designation of the location of the cotyledons on the central axis of the seedling. The general classification of hypogaeous or epigaeous was not adequate, especially where one anticipated continuous variation in the F_2 and other segregating generations. The method of measurement was to measure the distance from the cotyledons to the

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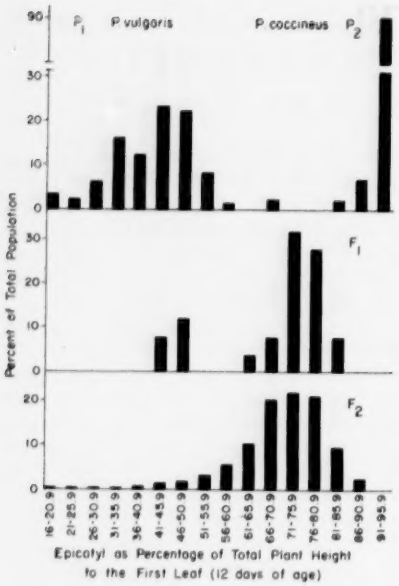

P. vulgaris
P. coccineus
Percent of Total Population
Epicotyl as Percentage of Total Plant Height to the First Leaf (12 days of age)

COTYLEDON POSITION

Figure 11

Frequency distributions of cotyledon position in *Phaseolus* seedlings in the parental lines and F_1 and F_2 generations.

first leaf and the distance from the cotyledons to the base of the central axis, the latter to be termed hypocotyl. These two values were reduced to one number, the epicotyl length being expressed as the percentage of epicotyl length plus hypocotyl length.

A greenhouse, where temperature and other factors could be controlled, was found to be the most satisfactory environment for investigating the mode of inheritance of cotyledon position. The seeds were scarified and planted in flats of steam-sterilized sand. The flats were placed on a heating cable in a greenhouse maintained at 75°F during the day and 65°F at night. On the 12th day after planting, the plants were pulled and measured. In order to avoid as much year-to-year variance as possible, the P_1, P_2, F_1, and F_2 were all germinated at one time, but, unfortunately, the F_3 and F_4 had to be germinated in a second planting at another time.

Results

The results presented in Table I and in Figures 11 and 12 appear to be substantial evidence for quantitative factor differences between *P. vulgaris* and *P. coccineus* for cotyledon position. If one major locus were responsible for the difference in cotyledon position and if one of the alleles were dominant, then the mean of the F_1 would be expected to coincide with the mean of one of the parents, and the F_2 distribution would be expected to be distinctly bimodal. As a matter of fact, however, the F_1 mean is 69.8 which is very near to being exactly intermediate between 41.8 and 93.2, the parental means, and the frequency distribution is not bimodal. Considering another monofactorial possibility, that of lack of dominance, there is no evidence of the tri-modality that would be expected in the F_2 distribution. Because of the smoothness of the distribution, especially in F_2, the likeliest explanation is that there are several different loci contributing to the difference between the two parents in cotyledon position. The amount of variability in cotyledon position among the three genotypes comprising the P_1 population is very suggestive that various genes are contributing to cotyledon position.

It may be said that the results are typically those of quantitative characters since:

1. The mean of the F_1 is approximately intermediate between the means of the two parents.

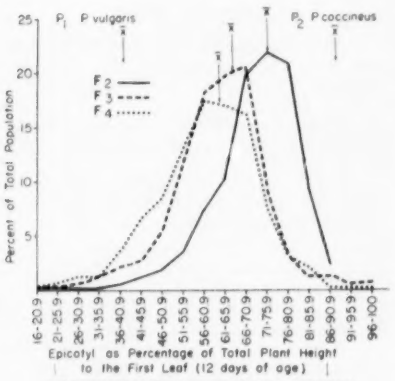
2. The means of the F_1 and F_2 are very similar.

3. The F_2 is appreciably more variable than the F_1, as shown by the spread of the histo-

TABLE I. Data on cotyledon position in *Phaseolus* seedlings

	Frequency Distributions																					
	Epicotyl % of total plant height to first leaf at 12 days of age																					
	16–20.9	21–25.9	26–30.9	31–35.9	36–40.9	41–45.9	46–50.9	51–55.9	56–60.9	61–65.9	66–70.9	71–75.9	76–80.9	81–85.9	86–90.9	91–95.9	96–100	N	$\bar{X}$	σ	$\sigma_{\bar{X}}$
P_1*	3	2	5	13	11	19	18	7	1		2							81	41.8	9.70	1.08
P_2*														1	3	39		43	93.2	2.17	.33
F_1						2	3			1	2	8	7	2				25	69.8	12.66	2.53
F_2	1	1	1	1	4	7	12	21	44	62	122	134	128	57	14			609	70.7	9.66	.39
F_3	1	1	3	8	14	19	35	79	121	132	138	60	22	8	9	4	6	660	62.3	11.16	.43
F_4	1	4	7	7	22	39	51	77	104	100	96	46	19	12	1	1	1	588	59.2	11.77	.49

*P_1 = *P. vulgaris*, P_2 = *P. coccineus*.



COYTLEDON POSITION SHIFT

Figure 12

Shift in cotyledon position in *Phaseolus* in the F_2, F_3, and F_4 of *P. vulgaris* × *P. coccineus* toward that of the *vulgaris* parent.

grams though this is not true for the σ values. However, the small size of the F_1 population and the known presence of one dwarf (see below) and the probability of others can account for this.

4. The extreme values of the F_2 measurements overlap well into the parental distribution though at a very small frequency in the more extreme classification intervals.

One anomaly within these data is that five of the F_1 plants fall in the frequency intervals 41-45.9 and 46-50.9. One of these plants was known to be a "physiological dwarf," such as often found in interspecific hybrids, and it is quite likely that the other four plants were also dwarfs, but since the plants were not grown beyond the 12-day stage, this supposition could not be verified. It is assumed that dwarfness may substantially modify cotyledon position by suppressing epicotyl growth.

The F_2 distribution is significantly skewed at the .01 level toward the *vulgaris* parent (Table II), and the extreme frequency interval of the *coccineus* parent does not appear in the population. However, in addition to including the most extreme *coccineus* parental

TABLE II. Measurement of F_2 and F_3 skewness by Pearson's method

F_2:

$$Sk = \frac{\bar{X}\text{–mode}}{\sigma} = \frac{70.7-73.5}{9.66} = -0.290$$

$$\sigma sk = \frac{1.225}{\sqrt{N}} = \frac{1.225}{\sqrt{609}} = 0.050^{**}$$

F_3:

$$Sk = \frac{62.3-68.5}{11.16} = -0.555$$

$$\sigma sk = \frac{1.225}{25.69} = 0.048^{**}$$

types, the F_3 and F_4 gave seven transgressive individuals in the 96-100 frequency interval. The hypocotyl length of these individuals was 0-3 mm compared to 8-12 mm for that of the *coccineus* parent. It appears that there is real skewing in the F_2 and F_3 generations.

We need to consider the skewness of the filial generations and what bearing it has on our explanation of the mode of inheritance of cotyledon position.

The skewness may be due to one of two things: to the dominance of the *vulgaris* alleles or to an increase in *vulgaris* alleles in the population from generation to generation. The first possibility seems unacceptable inasmuch as there was no evidence of dominance in the F_1. The latter possibility seems acceptable for the following reason: the more *vulgaris*-like plants tend to be more fertile than the more *coccineus*-like plants, they would therefore be able to build up the frequency of *vulgaris* genes in each succeeding generation.

Table I and Figure 12 illustrate the relationships between populations and the means of their parental types. In the F_2 the mean of the epicotyl as the percentage of epicotyl plus hypocotyl at 12 days was 70.7. The analogous mean value for the F_3 was 62.3 and for the F_4 59.2. Parent P_1 (*P. vulgaris*) has a mean value of 41.8 and P_2 (*P. coccineus*) has a mean value of 93.2. It is evident that there has been a large shift (8.4 percentage points) between the F_2 and F_3 means and that this shift is in the direction of the *vulgaris* parental type. Again between the F_3 and F_4 means there is a shift, though of less magnitude (3.1 percentage points), toward the *vulgaris* parent.

It is suggested that the explanation for the skewing of the F_2 in the direction of the *vulgaris* parent is the result of the elimination of gametes carrying high proportions of genes of *P. coccineus* and/or zygotic elimination of zygotes resulting from the union of gametes with a high proportion of *coccineus* genes. In this manner *coccineus* genes would be eliminated from the gene complex. It is to be remembered that we are dealing with *coccineus* genes functioning in *vulgaris* cytoplasm and that they may therefore be at a selective disadvantage. The progressive shift of the F_3 and F_4 toward the *vulgaris* parental type is consistent with such an interpretation.

The question arises as to how far cotyledon position might ultimately shift toward the *vulgaris* parent. It is logical to believe that there would be a continued but diminishing shift toward *P. vulgaris* in the F_3 and subsequent generations. It would depend partly on (1) how many *coccineus* genes influencing cotyledon position would ultimately remain in the population, and (2) the ultimate breeding behavior of the population, whether like *P. vulgaris* or *P. coccineus*.

It may be possible to devise a test which would give proof of gametic or zygotic elimination. Such a test would entail the use of a

marker gene from *P. coccineus* and a study of its subsequent behavior in the F_2, F_3, F_4, etc. Loss of the marker from the population would be corroborating evidence for gametic and/or zygotic elimination.

Summary

The position of the cotyledons on the central axis of *Phaseolus* seedlings is conditioned by multiple factors. There is no evidence of dominance. The frequency distribution for cotyledon position in the F_2 of *P. vulgaris* × *P. coccineus* is significantly skewed in the direction of the *vulgaris* parent. There are marked shifts in the frequency distributions for cotyledon position in the F_3 and F_4 toward that of the *vulgaris* parent. This may be explained on the basis of progressive *coccineus* gene elimination from the gene complex through gametic and/or zygotic elimination.

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THE X INTERNATIONAL CONGRESS OF GENETICS

The X International Congress of Genetics will be held at McGill University, Montreal, Canada, from August 20-27, inclusive, 1958. Congress Committees are now actively preparing for your arrival and raising funds for travel grants, particularly for overseas geneticists.

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If there is any possibility that you will be able to attend, we ask you to fill in the information requested below and send it to us *immediately*.

The Secretary General

Professor J. W. Boyes,
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McGill University,
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BRACHYURY IN COCKER SPANIELS

TERRY PULLIG*

A TYPE of abnormal development of the tail in cocker spaniels has been observed in two litters of puppies produced by the same male and female who were closely related. These deformed tails were all about one-half the length of a normal tail. Each short tail had at least one flexure but the breeder stated that in some cases there were several bends; thus, making docking by his method rather difficult. The remainder of the puppies in these two litters, as well as all the other puppies in three other litters whelped by this dam but by two other sires, were normal in every respect.

Variations in Tails Previously Reported in Some Mammals

A spontaneous dominant mutation, called Tail-short and assigned the symbol *T-s*, arose in the highly inbred strain C mice according to Morgan[3]. It was suspected that Tail-short behaved as a lethal in the homozygous state within strain C. When outcrossed to certain unrelated inbred strains the Tail-short mutation produced a new tailless phenotype.

According to Heston[1], a new mutation designated as vestigial tail appeared in the 68th brother-sister inbred generation of strain C56 brown mice. The tail was usually less than one cm. in length with approximately one-half the normal length being the extreme variation, and terminated in fused, irregular vertebrae.

Hollander and Strong[2] reported a dominant mutation, "pintail," which appeared in an inbred methylcholantrene-treated strain of mice. The principal feature was a reduction in the number of caudal vertebrae. This trait showed close linkage (five percent crossing-over) with brown.

According to Little[4], the allele for short tail in Brussel Griffons was dominant to its allele for long tail, and it produced a considerable degree of distortion in the short tails. There was reason to believe that this was an effect of the short-tailed gene and was not to be confused genetically with the tail kinks which are occasionally encountered in the long-tailed breeds.

When Stockard[6] crossed the screw-tailed bulldog with the long, straight-tailed basset hound, all the F_1 hybrids developed perfectly formed long tails. His results from the F_2 generation and the backcross to the pure bulldog stock indicated that there were two pairs of independent factors involved; that long and straight were the dominants, while short and bent were the recessive alleles, but that long dominated short more completely than straight dominated bent.

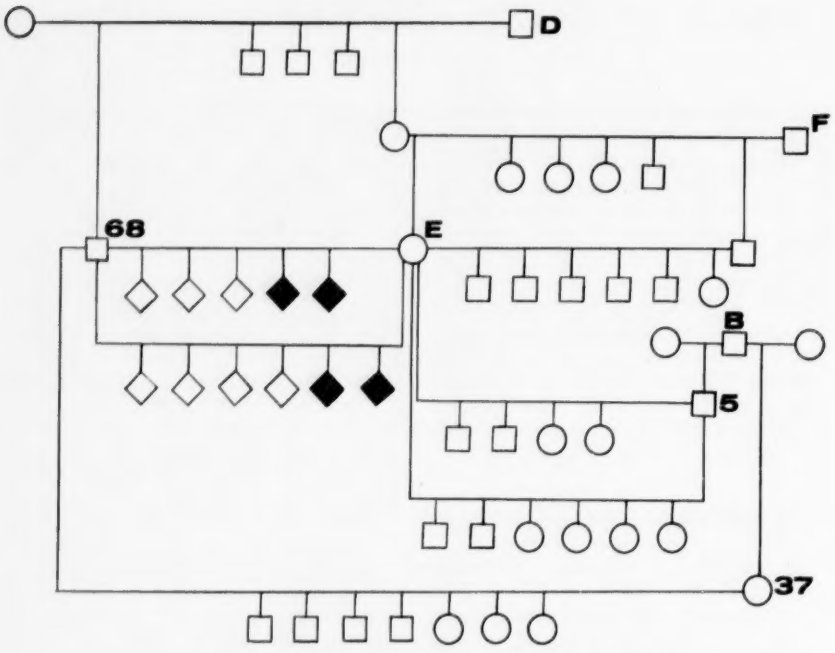
Pullig[5] reported that apparently there are two types of anury in the cocker spaniel; one, a sporadic type which is non-heritable and a second type which showed evidence that it is produced by a recessive gene.

Discussion

The mating of the daughter of male D to male F, whose great grandsire was the half-brother of male D, produced the female E and five litter-mates all with normal tails. Female E, the dam of all the short-tailed puppies, was first mated to her litter-brother and produced offspring all with normal tails. The second and third matings of the normal-tailed female E were to the normal-tailed male 68 who was the litter-brother of her dam. These two matings produced 11 offspring with each litter having two puppies with short, bent tails. The breeder could not recall the sex of each one in these two litters. For the fourth and fifth matings, female E was mated to male 5, who was related to her to the extent that they had common ancestors only in the 5th and 6th generations. The 10 puppies from these two matings did not show the anomaly. To date these five litters are all that female E has produced.

The normal-tailed female 37, the half-sister of male 5, was mated to the normal-tailed male 68 and all seven progeny had perfectly normal tails; giving further evidence that this short-tailed factor is recessive. Males 5, 68, B, and D and female 37 are the animals having these same numbers and letters in the paper on

*Aided by travel funds from the College of Arts and Sciences of Louisiana State University at Baton Rouge, Louisiana.



SHORT TAIL PEDIGREE

Figure 13

Inbred family of cocker spaniels in which short tails appear.

anury by Pullig[5]. The heritable type of anury was produced by male 5, one of his half-brothers, and a closely related male, when mated to the same closely related female. The author has seen male 5 and knows that he is not tailless. Based on the data reported by Pullig[5], male 5 must be heterozygous for the anury factor. But female E, who produced four short-tailed young out of 11 when mated to male 68, produced only normal-tailed puppies when mated to male 5 who had previously thrown taillessness; thus, indicating the probability that the factors for anury and brachyury are not alleles. A compilation of the data on anury and brachyury in cocker spaniels would seem to indicate that two different mutations have taken place in these two different inbred families; that these two anomalies are the result of two different recessive alleles. It is suggested that the following symbols be applied in cocker spaniels—*br* for brachyury and *an* for anury. In order for cocker spaniels to have normal tails the author's data is suggestive evidence that they must have at least two interacting genes; thus, there is no single factor for normal tail in cocker spaniels which has been indicated to be true for mice by other investigators.

Summary

The available data indicate that there is a recessive gene for brachyury in cocker spaniels and that this factor has a second effect on the tail by producing a varying number of flexures. The symbols *br* for brachyury and *an* for anury are suggested for these genes in cocker spaniels.

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X-RAY INDUCED TRANSLOCATION OF AGROPYRON STEM RUST RESISTANCE TO COMMON WHEAT

FRED C. ELLIOTT*

THE possibility of stabilizing valuable interchanges obtained through irradiation of interspecific or intergeneric F_1 hybrids and backcrosses is quite intriguing. The tendency for hybrids of this nature to segregate toward the parental types in the absence of recombination or interchange mechanisms is well known. Gamates carrying the chromosomes necessary to re-establish the parental types are more balanced and survive to unite with other such gametes. Irradiation of the hybrid seed or hybrid plant before meiosis may lead to interchanges. Upon fixation by selfing these interchanges can be propagated without the sterility commonly associated with heterozygous interchanges. In wheat, as in other self-fertilizing plants, homozygous translocations may be found in X_2. Subsequent segregation from a structural or genetic standpoint need not occur. At the present time there appears little hope of control over the induction of interchanges. However, the desired interchange may occur if large numbers of interchanges are induced.

Induced translocations offer possibilities in the transfer of characteristics, providing:

1. F_1 or BC_1 fertility is not seriously reduced;
2. Inheritance of the characteristic under transfer is relatively simple;
3. Genes or chromatin conditioning the characteristic act the same or similarly in the receptor selection as in the donor type;
4. Unfavorable pleiotropic effects are not involved; and
5. The critical genes or chromatin are not too closely linked with genes having deleterious effects on growth, reproduction, and adaptation.

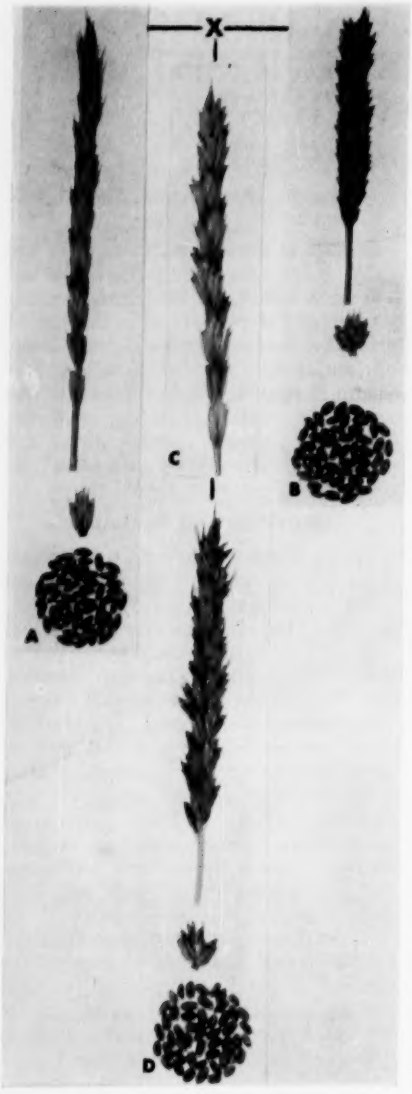
In spite of these restrictions there are many applications to be made in the improvement of crop plants. Sears[6] recently outlined the transfer of resistance to leaf rust (*Puccinia triticina*) from *Aegilops umbellulata* to common wheat. The example reported here involves the transfer of resistance to wheat stem rust (*Puccinia graminis tritici*) from tall wheatgrass, (*Agropyron elongatum*), to a hexaploid, common wheat.

Materials and Methods

The first step was to cross a tall wheat grass ($2n = 70$) to a hexaploid wheat variety. This was accomplished by W. J. Sando at Beltsville, Maryland, in the late 1930's. The hybrid seeds were sent to various Experiment Stations, including the Washington Agricultural Experiment Station at Pullman, Washington. Stem rust resistance was stabilized in octoploid ($2n = 56$) derivatives after a few segregating generations in the Pullman nursery[5]. These derivatives were winter types intermediate in morphology between the parents. The culms and leaves were quite harsh and wiry and the caryopses were shriveled.

A resistant octoploid derivative as the female parent was next crossed to the susceptible hexaploid spring variety, Idaed. The dry, hybrid seeds ($2n = 49$) = (F_1 dormant embryos) were then irradiated in various ways, (see Figure 14 and also Larter and Elliott[2]). The X_1 and control populations were space planted in the nursery at Pullman. The X_2's as well as X_3 progeny rows from resistant X_2 plants were screened in the rust nursery of the Dominion Cereal

*Associate Professor, Agronomy Department, State College of Washington. Scientific Paper No. 1582. Washington Agricultural Experiment Stations. Work conducted under Project No. 966.



PARENTAL AND IRRADIATED TYPES

Figure 14

Parental, X_1, and X_5 spikes, spikelets, and caryopses of the parents and a rust-resistant X_5 derivative. *A*—SH 198-4-5, ($2n = 56$); *B*—Idaed, ($2n = 42$); *C*—X_1, ($2n = 49$); *D*—Sel. 11244, X_5, ($2n = 42$).

Breeding Laboratory, Winnipeg, Manitoba.

In the 1954 Winnipeg nursery one rust-resistant, wheat-like X_2 plant was found in an 18,000r X-ray population. The remaining plants in this population were susceptible with the exception of two, weak, sterile grass-like plants. The glumes of the resistant, wheat-like plant were red, in contrast to the white glumes of both parents.

Red glume color in hexaploid wheats is conditioned by a dominant factor or factors (see 1946 Majority Committee Report on Nomenclature of the American Society of Agronomy[1]). In the case of the variety Federation 41, this factor has been assigned to chromosome 1 of Chinese Spring by Unrau[7]. The glumes on X_1 plants of the cross shown in Figure 14 were white in all recorded observations. The X_3 row from this red-glumed, rust-resistant X_2 plant was uniformly red-glumed and rust-resistant. No obvious segregation for glume color or other morphological characteristics was observed in an X_3 row from remnant seed, in 62 X_4 progeny rows, or in 25 X_5 progeny rows grown simultaneously in the 1956 Pullman nursery. Under conditions in the 1956 Pullman nursery the leaves of the rust-resistant stock remained green after the culms turned a golden color, presenting a striking contrast not seen in ordinary wheat varieties approaching maturity.

Pollen-mother-cells of several progeny in each row were taken for cytological analyses. Crosses of the rust-resistant stock in both the X_4 and X_5 were made again to Idaed during the flowering season of 1956. A number of the F_1's were grown in the greenhouse at Pullman in the winter of 1956-57 and PMC's of F_1 hybrids and parents were again taken. Spikes were taken from greenhouse plants after four to five hours of sunshine and fixed in Newcomer's solution[4]: 6 parts isopropyl alcohol; 3 parts propionic acid; 1 part petroleum ether; 1 part acetone; 1 part dioxane. The material was stored in the refrigerator about three weeks at 40° F. After re-

METAPHASE IN F_1 HYBRID

Figure 15

A—Metaphase I in F_1 hybrid (101-26-6 × Idaed) showing: three univalents, one open bivalent, 13 closed bivalents, one trivalent and two quadrivalents. *B*—Metaphase I in another PMC of the same anther shown in *A* with two univalents, four open bivalents, and 16 closed bivalents.

moval from the refrigerator, the spikes were placed in 45 percent propionic acid and individual florets were removed under the dissecting microscope. Individual anthers were cut up and smeared in propionic carmine; no heat was applied to the slides.

Results

No appreciable differences were observed in the metaphase I associations of plants of the rust-resistant stock (SH 198-4 × Idaed, 18,000r X-ray treatment) within or between generations (see Table I). These derivatives were fairly well stabilized at a $2n = 42$ chromosome number with but few exceptions, as noted in Table I.

A summary of the metaphase I associations in the F_1 hybrids between the rust-resistant stock and Idaed grown in the greenhouse during the winter of 1956-57 is presented in Table II.

Photomicrographs showing Metaphase I associations in the F_1 hybrids and in one of the rust-resistant parent lines are shown in Figures 15*A* and *B* and 16.

The number of micronuclei were counted in 100 quartets of each of three F_1 hybrids. The mean percentages of quartets with 0 to 3+ micronuclei per quartet are presented in Table III.

Discussion

At least three multivalent associations were observed in the same cell of an F_1 hybrid (rust-resistant stock × Idaed). Therefore, one might assume that at least three interchanges were involved in differentiating the karyotype of the rust-resistant stock from Idaed. The increased number of open bivalents and infrequent occurrence of multivalents in the F_1 hybrids suggest that the possible translocations were small (presumably intercalary) and that chiasma formation was inhibited. One might also speculate that since an association of six chromosomes was seen in individual PMC's from two different F_1 hybrids that one pair

TABLE I. Metaphase I associations in PMC's of rust-resistant derivatives (SH 198-4 × Idaed 18,000r X-ray) grown in the 1956 Pullman nursery

Generation	No. of Progenies Examined	Total No. of Plants Examined	No. of PMC's		I	II open	II closed	IV	$2n=$
X_3		6	34	mean	..	.9	20.0	.03	42
				range	..	0-4	17-21	0-1	
X_4*	62	206	989	mean	.11	1.01	19.85	.015	42
				range	0-4	0-4	17-21	0-1	
X_5†	25	82	391	mean	.12	.9	19.97	.04	42
				range	0-3	0-5	16-21	0-1	

*Five plants with fragments, one plant with one isochromosome, and three monosomic plants were found in a total of 206 X_4 plants.

†One plant with one isochromosome and one monosomic plant were found in a total of 82 X_5 plants.

RUST RESISTANT X_5
Figure 16

Metaphase I in a rust-resistant X_5 parent. 101-26-6 with one open bivalent, and 20 closed bivalents.

of chromosomes involved have at least two translocated segments. The evidence of interchanges observed in these hybrids, of course, does not imply that only translocations of wheat chromosomes with extra chromosomes from the octoploid parent were involved. Later studies may reveal that rearrangements within the original hexaploid wheat complement have also taken place.

The red glume color may have resulted from the deletion of inhibitors, suppressors, or modifiers in one or both of the parents, or as a position effect similar to the effect associated with yellow-green seedlings in barley irradiated in the Bikini tests and discussed by Moh, Nilan, and Elliott[3]. The induction of a dominant mutation for red glume color was ruled out, since a simultaneous change in the same locus of both homologues would be required to avoid segregation. Volunteer plants of this type in the Winnipeg nursery were eliminated as a source of the original red-glumed, rust-resistant selection. Contamination from outcrossing to red-glumed varieties in the X_1 generation at Pullman was also eliminated as a source of the characteristic since segregation failed to occur in succeeding generations.

Selections comparable to the rust-resistant stock have not been detected in the Pullman nurseries among large numbers of segregating control (non-irradiated) populations.

This material has shown a high type of resistance to the races prevailing in the rust nursery at Winnipeg (including numerous biotypes of race 15B). Thus, it should prove to be an interesting source of stem rust resistance, particularly when recombined with other available sources of resistance to both stem rust and leaf rust. In view of the irregular pairing at metaphase I and the chromatin excluded from the main nuclei of the microspores, future genetic studies of this source of rust resistance should be accompanied by cytological analyses. The material under discussion is not expected to be used directly as a variety. This material lacks certain requisites for milling and baking quality. It is, in addition, susceptible to leaf rust.

Summary

The translocation of resistance to wheat stem rust displayed by an octoploid derivative involving *Agropyron* elongatum to a hexaploid wheat has been achieved through irradiation. At least three interchanges differentiate the karyotype of the rust-resistant stock and the susceptible parent variety Idaed. Rearrangements within the hexaploid wheat complement may also be involved.

The rust-resistant stock has red glumes, a dominant condition not present in either parent. This condition may have arisen as a result of deletions affecting modifier, suppressor, or inhibitor relationships in one or both parents accompanying the translocations, or as a position effect.

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TABLE III. Mean percent of quartets with various numbers of micronuclei

No. of micronuclei	Mean % of quartets
0	71.3
1	11.0
2	9.0
3	7.3
3+	1.3

TABLE II. Metaphase I associations in the F_1 hybrids between the rust-resistant lines and Idaed and in one of the parent plant lines

	No. of Plants	No. of PMC's	I	II			III	IV	VI	$2n=$
				open	closed	total				
F_1 hybrids	6	47	mean 1.02	3.6	16.0	19.6	.085	.255	.043	42
			range 0-8	0-7	8-21	14-21	0-2	0-2	0-1	
Rust Resistant Parent	1	5	mean	1.4	19.6	21.0	–	–	–	42
			range	0-4	17-21	21				

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◆

EVIDENCE OF A NEW LETHAL GENE IN CATTLE

P. R. Ternan, J. F. Kidwell and L. Walker*

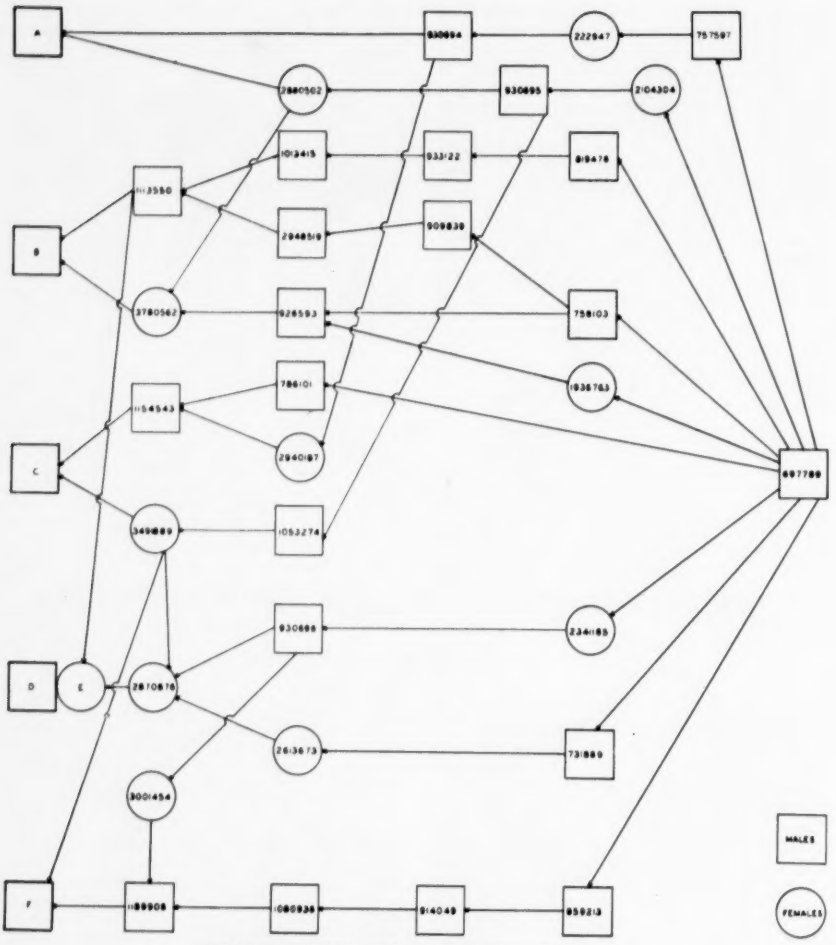
In most dairy herds some calves are born dead at normal term, or die soon after birth. Many can be accounted for on the basis of calving difficulties, accidents at parturition, pathological or other known causes. Some, however, cannot be explained by known causes. Analysis of the records of calves born dead, or which died shortly after birth in the University of Nevada purebred Holstein-Friesian herd during the period 1953-1956, suggests the existence of a recessive autosomal lethal gene. The purpose of this note is to present the available evidence.

All calves born in the herd during the period 1953-56 are classified in Table I. Six of the 11 calves in the classification "born dead or died within six days" exhibited a number of common symptoms. These six calves are shown in the pedigree network of Figure 17. The numbers are Holstein-Friesian Association registration numbers.

These calves were all born alive with normal delivery. One lived six days, another five, and the others from one to three hours after birth. They exhibited a very rapid heart beat and rapid, labored breathing. The heart sound was also abnormal. The two that lived more than a few hours remained in a state of torpor. They were under the supervision of a practicing veterinarian from about three hours after birth until they died. He reported no fever or other indication of pathological infection. The attending veterinarian concluded that the symptoms were quite indicative of a congenital circulatory defect and suggested that patent foramen ovale or patent ductus arteriosus might produce the observed symptoms. These calves were not autopsied.

Only one calf has subsequently been available for study. It died about thirty minutes after birth. Autopsy by the University veterinarian indicated a patent foramen ovale, but no other gross morphological defects were observed. Sisson and Grossman[2] indicate that the foramen ovale is normally open at birth, but soon closes. A small part of the foramen may persist in the adult without apparent disturbance of the circulation. Boyd[1] indicates that the ductus arteriosus is patent at birth but obliterated at three to four weeks. The present anatomical description and analysis are incomplete and insufficient to warrant any conclusions. However, it seems quite possible that a circulatory defect involving the morphology or physiology of the foramen ovale and/or ductus arteriosus may be responsible for the early postnatal deaths.

*Animal Husbandry Department, University of Nevada, Reno.



RELATIONSHIP OF AFFLICTED CALVES

Figure 17

Pedigree network illustrating relations among afflicted calves. Caution must be used in interpreting this as evidence of an autosomal recessive gene. One hundred thirty-seven of the calves born in the herd during the period of this study trace trough sire and dam to bull number 697789.

All of the afflicted calves trace through both sire and dam to male number 697789, Wisconsin Admiral Burke Lad (Figure 17). This suggests the existence of a simple autosomal recessive gene. This evidence, however, is far from conclusive and perhaps quite misleading. One hundred and thirty-seven of the 149 calves born during the period of this study trace through both sire and dam to this bull. There is no evidence of sex linkage or sex influence. There are insufficient data for statistical analysis.

Although the foregoing physical description is inadequate and the genetic evidence meagre, it suggests the existence of a heretofore unreported lethal gene. It is hoped that this brief note will serve to call the attention of other workers to the problem. Additional data are required to verify or disprove the existence of a recessive autosomal gene conditioning a

circulatory anomaly in cattle. Actual registration numbers are used in Figure 17 to assist investigators who may study related animals.

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TABLE I. Classification of calves born in University of Nevada Purebred Holstein-Friesian herd during 1953-56 inclusive

	Calves born alive and lived at least 7 days		Calves born dead or died within 6 days				
			Cause Known			Cause Unknown	
Year	Male	Female	Male	Female	Sex Undetermined	Male	Female
1953	14	15	0	0	0	2	0
1954	14	24	0	0	0	3	0
1955	11	19	0	0	4	4	1
1956	13	21	3	0	0	1	0
Total	52	79	3	0	4	10	1

Research Proposals in the Life Sciences Announced by the National Science Foundation

The Division of Biological and Medical Sciences of the National Science Foundation announces that the next closing date for receipt of research proposals in the life sciences is September 15, 1957. Proposals received prior to that date will be reviewed at the Fall meetings of the Foundation's Advisory panels and disposition will be made approximately four months following the closing date. Proposals received after the September 15, 1957 closing date will be reviewed following the Winter closing date of January 15, 1958.

In addition to funds for the support of basic research in the life sciences, limited funds will bs available during Fiscal Year 1958 (July 1, 1957—June 30, 1958) for the support of research facilities and programs at biological field stations.

Inquiries should be addressed to National Science Foundation, Washington 25, D. C.

A QUANTITATIVE GENETIC STUDY OF A NUTRITIONAL STRAIN DIFFERENCE

In Mice

MERTON S. HONEYMAN*

THE nutritional requirements of mice and rats have been studied by numerous investigators. Some workers have reported that strains of mice differ with respect to their quantitative requirements for certain nutrients and in their response to the same nutrients[3, 4, 5, 6, 8]. Although inbred strains of mice were used in the experiments mentioned above, no genetic investigations of nutritional strain differences have been reported. This paper deals with such a genetic investigation.

Materials and Methods

Male mice of the C57BL/10 and BALB/c strains were fed a 10 percent protein diet to determine if there was a strain difference in weight gain. Only male mice were used, in order to eliminate any differences in weight gain due to sex. Six hundred and fifty-four male mice of the C57BL/10 and BALB/c strains (P_1 and P_2 respectively) and of the first (F_1) and second (F_2) filial generations and the first (B_1 and B_2) and second (B_{11}, B_{12}, B_{21}, B_{22}) backcross generations were reared on the 10 percent protein and control diets. The mating procedure described by Green[7] was used to obtain the F_1 and F_2 and the B_1 and B_2 generations. The number of mice in each generation are listed in Table I.

All the BALB/c mice were in the 48th to 53rd generation of inbreeding by brother-sister matings, and were descended from two pairs, one in the 47th generation and the other in the 49th generation of brother-sister matings. The mice of the C57BL/10 strain were in the 42nd to 44th generation of brother-sister inbreeding and were descended from a single pair in the 40th generation. The mice were obtained from the mouse colony maintained at The Ohio State University by Dr. E. L. Green.

The mice were weaned at 21 days of age, weighed, and placed on the 10 percent protein diet or on the control diet *ad libitum*, until they were 51 days of age. The mice were weighed every second day to the nearest tenth of a gram on a direct reading shadograph scale, model 4104B.

The 10 percent protein diet used was diet 241 of Fenton and Carr[5] with a slight modification of the trace elements; 0.9 grams of $CuSO_4$ and 0.34 grams of borax were used in place of 0.5 grams of $CuBO_4$. The control diet used was Purina Laboratory Chow, which contains 26.8 percent protein. The 10 percent protein diet was utilized for the major portion of the experiment as it was possible to control the source of its components.

The weight gains were collected over a period of ten months (July 2, 1953-April 20, 1954). The weight gains were affected by seasonal variation, the weight gain of the mice being at a maximum during the cooler seasons and at a minimum in the warmer seasons of the year. The effect of seasonal variation was diminished by fitting the observed weight gains to the sine curve $Y = A\sin(X + \bar{\phi} + L)$,

where Y is the expected weight gain for day X,
A is the amplitude of the curve,
X is the day the mouse started on the diet, measured in degrees,
$\bar{\phi}$ is the average phase angle of the two parental strains, and
L is the estimate of the mean. The

*Department of Zoology and Entomology, the Ohio State University, Columbus. Present address: Biometry Section, National Cancer Institute, National Institutes of Health, Bethesda, Maryland. Based up a dissertation presented to the Faculty of the Graduate School of The Ohio State University in partial fulfillment of the requirements for the Ph. D. degree.

adjusted weight gain for the individual was obtained by $P = O - Y + L$,

where P is the adjusted weight gain of the individual,

O is the observed weight gain of the individual,

Y is the expected weight gain of the individual,

and L is the estimate of the mean. (The observed and adjusted mean weight gains for the various generations are shown in Table I.)

The sine curve adjustment has been used by Yoon[12] in the removal of the effects of seasonal variation. In this instance, the use of the sine curve was determined by the method of least squares.

Results

The weight gain on the 10 percent protein diet of sublines within the two parental strains were compared to determine whether the parental strains were homogenous. No difference in the weight gain within the two parental strains was observed. Nor was there any difference found in the weight gain of F_1 individuals resulting from reciprocal crosses. The mean weight gain of F_1 individuals with C57BL/10 mothers was 10.67 ± 0.40 grams, and from individuals with BALB/c mothers 10.53 ± 0.63 grams.

A comparison of the mean weight gains of the P_1 and P_2 generations on the 10 percent protein diet and on the control diet revealed that there was a difference in weight gain between the two strains on the control diet, as well as a highly significant difference of 2.6 grams in weight gain on the 10 percent protein diet. (The phase angles, ϕ_1 and ϕ_2 of the P_1 and P_2 generations were used in obtaining the means utilized in this comparison instead of the common phase angle, $\bar{\phi}$, which would have shown a significant difference of 2.8 grams for those animals reared on the 10 percent protein diet.) The comparison of the mean weight gains on the control diet and the 10 percent diet are shown in Table II.

Mather[9] has shown that scaling is extremely important. A scale is adequate when the genic effects are additive and the contributions to weight gain by the non-heritable agents are independent of the genotype. The criterion of a difference used was weight gain in grams. Therefore, it was necessary to determine if the observations could be used in any or all of the crosses as measurements in grams or whether they must be transformed to another scale.

The symbols and statistics of Mather[9] for testing the adequacy of scale were used. The constants A, B, C, D, E, and F did not show significant deviations from the expected value of zero. However, G differed significantly from the expected value due to the excessive value of the B_{B1} mean. The B_{B1} mean was excessively high, this may be due to the fact that the B_{B1} observations were taken at a period corresponding to the maximum point on the sine curve used to adjust the data, and slight variations from the expected tended to inflate the adjusted mean. Therefore, this deviation was ignored and the scale deemed to be adequate. The results of the test scale are tabulated in Table III.

The components of variation can be divided into the heritable and the non-heritable components[9]. The latter results from the action of the environment and this is designated by the symbol E. The heritable components of variation result from the activity of the genes controlling the character involved.

In the present experiment, the non-heritable variation E can be divided into the variation due to seasonal effects and the variation due to environmental factors other than seasonal variation. It is assumed that the majority of the seasonal variation has been removed by fitting the data to a sine curve and, therefore, E will represent only the environmental effects other than seasonal variation.

TABLE I. Number of males observed in the different generations on diet 241 and on the control diet, and the observed and adjusted mean weight gains of the different generations on diet 241

Generation	Number of Males		Mean Weight Gains (grams)	
	Diet 241	Control Diet	Observed	Adjusted
P_1	81	26	8.08 ± 2.86	8.17 ± 0.208
P_2	80	24	10.92 ± 2.26	10.96 ± 0.206
F_1	52		7.94 ± 2.58	10.57 ± 1.27
F_2	88		11.24 ± 2.70	10.27 ± 0.319
B_1	54		10.84 ± 2.76	9.15 ± 0.515
B_2	57		11.77 ± 2.23	11.42 ± 0.294
B_{11}	49		11.51 ± 2.34	9.56 ± 2.66
B_{12}	50		12.33 ± 1.77	10.29 ± 1.87
B_{21}	42		13.05 ± 2.09	12.00 ± 2.36
B_{22}	51		13.06 ± 1.62	17.08 ± 8.74
Total	604	50		
Grand Total	654			

The non-heritable component of variation is divided into two classes in the present experiment, E_1 and E_2. E_1 represents the variation within sibship groups and E_2 represents variation between sibship groups. Since the P_1, P_2, and F_1 generations are genetically alike within each group, the variations of these generations can be taken as direct estimates of the environmental variation. Although no difference existed in the variances of the P_1, P_2, and F_1 generations, the variances of the reciprocal groups of the F_1 generation were 7.97 (BALB/c mother) and 4.81 (C57-BL/10 mother), ($F=1.66$, $F_{(.05,20,30)}=2.04$). In view of this difference in the variances of the F_1 generation even though they are not significantly different, it was deemed advisable not to make a direct estimate of E_1 and E_2 from the observed data.

The heritable variation may be divided into fixable and unfixable variations. Mather[9] describes the fixable variation as due to the differences in the average character expression associated with the two homozygotes for each pair of genes involved. The unfixable variation arises from the differences between the expression of the heterozygotes and the average of the two corresponding homozygotes. The magnitude of the unfixable variation is dependent on the dominance relation of the genes.

The deviation of the two homozygotes of a pair of genes from the midpoint or average may be represented by +d or −d, and the deviation of the heterozygote from the midpoint may be represented by h. Therefore, the contribution of the homozygotes to the sum of squares is $S(+d^2)$ and the heterozygotes is $S(h^2)$ for the heterozygotes. These may be conveniently denoted by the symbols D and H respectively.

The variances, mean variances, and variance of the means of the crosses were obtained by utilizing the formulae of Mather[9] as follows:

$$V_{F_2} = \tfrac{1}{2}D + \tfrac{1}{4}H + E_1 = 5.78$$

$$V_{B_1} + V_{B_2} = \tfrac{1}{2}D + \tfrac{1}{2}H + 2E_1 = 9.84$$

$$\overline{V}_{B_{11}} + \overline{V}_{B_{12}} = \tfrac{1}{4}D + \tfrac{1}{4}H + 2E_1 = 5.49$$

$$\overline{V}_{B_{21}} + \overline{V}_{B_{22}} = \tfrac{1}{4}D + \tfrac{1}{4}H + 2E_1 = 5.51$$

$$V_{\overline{B}_{11}} + V_{\overline{B}_{12}} = \tfrac{1}{8}D + \tfrac{1}{8}H + 2E_2 = 5.55$$

$$V_{\overline{B}_{21}} + V_{\overline{B}_{22}} = \tfrac{1}{8}D + \tfrac{1}{8}H + 2E_2 = 3.96$$

The following estimates were obtained when solving the above equations for D, H, E_1, and E_2 by the method of least squares as described by Mather[9]:

$$D = 3.53 \pm 1.43$$
$$H = 13.65 \pm 2.47$$
$$E_1 = 0.607 \pm 0.279$$
$$E_2 = 1.307 \pm 0.151$$

As D depends on differences between individuals distinguished by being homozygous for the two alleles of the various genes, and H depends on the departure of the heterozygote from the mean of the two comparable heterozygotes, then the value of D obtained indicates the existence of a definite genetic difference in the two parental strains for the character investigated and the value of H obtained being significantly larger (10.12 ± 2.86) than the value of D is indicative of heterosis.

TABLE II. Comparison of weight gain of the P_1 and P_2 strains on diet 241 and the control diet

Diet	Strain	Mean	Variance	df	t	t(.01)
Control	P_1	14.51	1.32	48	8.60	2.678
	P_2	12.11	0.701			
241	P_1	8.17	3.32	159	9.62	2.605
	P_2	10.96	3.59			
Control	P_1	14.51	1.32	105	16.86	2.624
241	P_1	8.17	3.32			
Control	P_2	12.11	0.701	102	2.89	2.626
241	P_2	10.96	3.59			

Discussion

Evidence has been presented which shows that the C57BL/10 and BALB/c strains of mice differ in their mean weight gain on a 10 percent protein diet, and on a control diet containing 26 percent protein. Searle[11] has reported that the feeding of a poor diet which slows growth and metabolism decreases the mean weight of C57BL/gr mice and inflates the variance of the weight of these mice. A decrease in the mean weight gain was found on the 10 percent protein diet compared with the mean weight gain on the control diet, and the decrease in the mean weight gain was accompanied by an inflation of the variance.

The quantitative inheritance of weight gain indicated is in agreement with the studies of

TABLE III. Test of scale

Quantity	Deviation From Zero	Standard Error	Test of Significance t	Probability
$A = 2B_1 - P_1 - F_1$	−0.44	4.45	−0.988	P 32%
$B = 2B_2 - P_2 - F_2$	1.31	3.37	0.388	P 70%
$C = 4F_2 - 2F_1 - P_1 - P_2$	0.81	5.63	0.144	P 88%
$D = 2B_{11} - B_1 - P_1$	1.80	4.25	0.424	P 67%
$E = 2B_{12} - F_1 - B_2$	−1.41	3.62	−0.389	P 70%
$F = 2B_{22} - F_1 - B_1$	4.28	5.33	0.803	P 42%
$G = 2B_{22} - B_2 - P_2$	11.79	2.34	5.03	P < 1%

several authors who found that body size and weight are quantitatively inherited in ducks, maize, and mice[1,2,10]. As indicated by observation of the adjusted weight gains presented in Table I, the data supported the presence of heterosis.

The present experiment shows that a nutritional difference exists between two inbred strains of mice and the difference is of a quantitative genetic nature.

The implications of these findings are that there are other nutritional differences existing between inbred strains of organisms which would bear investigation, an example of which are the differences found by Fenton and his coworkers in the nutritional requirements of different strains of mice.

Summary

A difference in weight gain on a low protein diet between two inbred strains of mice C57BL/10 and BALB/c was studied by genetic analysis of gain in weight from 21 to 51 days of age of the parental, F_1 and F_2, and B_1 and B_2 generations.

Sublines of the parental strains were homogenous. Therefore, it is assumed that the parental strains were homozygous at all loci affecting weight gain.

A significant difference existed in the mean weight gains of the parental strains on the low protein diet and on the control diet.

No difference was found in the mean weight gain of reciprocal F_1 individuals. Therefore, it was assumed that no maternal influence was involved.

The assumptions of additivity of genic effects and independence of environmental agents and genotype were supported.

The weight gain of the inbred strains of mice was found to be controlled by quantitative genetic factors, and heterosis was indicated.

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THE PRODIGAL RETURNS!

NIKITA S. Khrushchev, Soviet Communist party secretary, has thrown his weight behind Trofim D. Lysenko, controversial Soviet agronomist, in a major dispute over farming techniques.

Mr. Khruschev has given strong public support to Mr. Lysenko in a dispute among Soviet agricultural experts over the best methods of mixing organic and mineral fertilizers.

Mr. Khrushchev's gesture did not seem to indicate that Mr. Lysenko was to resume the post of scientific ideological czar that he held under Stalin. But it did serve to re-establish Mr. Lysenko's reputation in the field of practical agriculture.

The Khrushchev action occurred at an agricultural meeting held in Moscow last week and reported in Moscow newspapers reaching New York.

Ministers in Dispute

The Moscow radio, according to the United Press, reported yesterday that Mr. Khrushchev had criticized Vladimir V. Matskevich, Minister of Agriculture, and Ivan A. Benediktov,

Minister of State Farms, for "sitting by with their hands folded like saints" in the controvesy over Mr. Lysenko's technique of mixing organic and mineral fertilizer.

Mr. Khrushchev in a speech at Gorky on Monday was said to have charged that members of the Academy of Agricultural Science employed "police" methods to defeat Mr. Lysenko's program.

"Why do some scientists oppose Lysenko?" Mr. Khrushchev asked. "Some scientists went to great lengths to accuse Lysenko of all sins when he was president of the academy. That is why we agreed when he asked to be relieved of his post."

In Moscow last week Mr. Khrushchev disclosed that he had urged Mr. Lysenko to speak at the meeting on the subject of fertilizers. Then, too, the party leader criticized Mr. Lysenko's foes.

Voices Faith in Lysenko

"There are scientists," Mr. Khrushchev said, "who still argue with Lysenko on this question. If you asked me for which scientist I would vote, I would say without hesitation—for Lysenko. And I know that he will not let me down because he does not untake anything poor. I believe that few of our scientists understand the soil as does Comrade Lysenko."

Mr. Khrushchev said he had visited Mr. Lysenko's experimental farm three years ago and was convinced that the scientist's technique produced excellent results.

The public tribute to Mr. Lysenko by Mr. Khrushchev modified the criticism to which Mr. Lysenko was subjected after the death of Stalin.

Mr. Khrushchev himself was a leader in the criticism of Mr. Lysenko at that time.

Mr. Khrushchev accused Mr. Lysenko then of falsifying experiments in the interests of supporting his dubious genetic theories and of attempting to set up a dictatorship in the field of science.

HARRISON E. SALISBURY

The New York Times,
April 11, 1957

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